ARCHIVES OF OPHTHALMOLOGY.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF RETINAL HEMORRHAGES.

By Dr. J. THALBERG, St. Petersburg, Russia.

Translated by Dr. A. Schapringer, New York.

THE following was a case of retinal hemorrhage, due to anæmia caused by caseous degeneration of the bronchial and mesenteric glands, and acute miliary tuberculosis of the lungs. The picture of the disease bore such a marked resemblance to progressive pernicious anæmia that the patient was at first thought to be suffering from the latter complaint. The ophthalmoscopic picture received a satisfactory explanation by the post-mortem examination. The result of the latter, as far as the retinal hemorrhages went, was similar to that hitherto obtained in cases of progressive pernicious anæmia.

Alexei K., seventeen years old, the son of a soldier, was admitted to the Semenow Military Hospital on September 18, 1882, with the diagnosis of progressive pernicious anæmia. The anæmic condition was well marked, the mucous membranes being extremely pale, but there was no emaciation. There were great debility and dyspnœa. The patient was distressed by a hacking cough. His temperature was increased every evening. Physical examination disclosed nothing but the evidences of slight chronic catarrhal inflammation of both apices, in the left lung more than in the right. The other organs were healthy. There was no enlargement of the spleen or the lymphatic glands. The bones were not tender on pressure. The ratio of white and red blood corpuscles was as 1 to 50.

The boy's general condition became gradually worse. The cough persisted. Dry as well as moist râles made their appear-

ance in the apices of the lungs. His temperature in the evening rose to or even exceeded 104°. At night he would become delirious, and later on his morning temperature also rose above the normal.

He began to complain of his eyesight, saying that objects in the daytime appeared to him suffused by a pink light, and in the evening the light of the lamp seemed to have a dark-red color. An ophthalmoscopic examination being made on October 13th, the optic discs were found to be pale and somewhat swollen, with blurred outlines. The retinal vessels, especially the veins, were distended and tortuous. Around the optic disc of either eye were seen striated hemorrhages of a bright-red color, in a stellate arrangement, and running parallel to the blood-vessels. In some places the vessels were completely ensheathed by these extravasations, whilst in others the spaces between the vessels were completely occupied by dark-red hemorrhages, the boundaries of which were formed by the vessels themselves. At some distance from the disc the size of the striated extravasations diminished, and at the equator they appeared only as dark-red dots in front of or near the blood-vessels. The space between the optic-nerve entrance and the macula lutea in both eyes was occupied by a large hemorrhagic patch of oval shape. Several punctiform, shining, white dots were scattered among the extravasations above the disc. They were more numerous in the left eye, in which the number and size of the hemorrhages were also greater. Within the area occupied by the extravasations the retina was gray opaque, and swollen, the change being most marked at the margin of the hemorrhagic patches. The peripheric parts of the retina also showed a pale-gray discoloration, but without opacity. The refracting media were clear. On account of the extreme debility of the patient, the condition of the fields of vision could not be determined. The acuteness of vision of the right eye was $=< \frac{20}{50}$, that of the left $= \frac{8}{200}$.

On the day of this examination the patient was taken with pneumonia of the left lower lobe, with high fever, and he expired on October 15th.

At the **autopsy** the pericardium and both pleural cavities were found filled with serum. The lower part of the left lung was hepatized, the rest of the lung tissue was consolidated. The surface of both lungs was dotted by a large number of miliary tubercles. The bronchial and mesenteric glands contained caseous masses.

The right eyeball only was removed, and opened by a cut placed in the sagittal plane. A dark-red, oval patch of hemorrhage was found to occupy the space between the optic-nerve entrance and the macula lutea. The patch was traversed in a horizontal direction by a gray fold of swollen retinal tissue. A similar fold encircled the hemorrhage. The retina surrounding the optic-nerve entrance was swollen, and studded with striated extravasations, which exhibited a stellate arrangement. These extravasations accompanied the blood-vessels, and were placed on either side of them. They became smaller toward the periphery of the retina, and could not be traced beyond the equator. Two dark-red dots marked the place of bifurcation of the central retinal vessels. The shining white spots found on ophthalmoscopic examination could not be recognized.

Microscopic Examination.—The optic nerve did not show any pathological changes, and there were no hemorrhages along its blood-vessels. The changes began only beyond the region where the bundle of nerve-fibres arch over the disc into the retina and the bifurcation of the vessels. All around the optic-nerve entrance the bundles of nerve-fibres were thickened and loosened, with wide, empty spaces between them. The elevation seen macroscopically upon and around the large central hemorrhagic patch was found to consist mainly of a thickened fold of the nerve-fibre and ganglionic layers; the underlying layers of the retina, including the pigment epithelium, being only slightly raised. The vessels of the diseased portion of the retina were considerably distended, especially the capillaries and the veins. In some places all the layers lying beyond the ganglionic layer were pushed outward toward the choroid by a distended vein ; in others the layers were crowded asunder, the rent thus formed being filled by a finely granular mass with some sparse white and red blood corpuscles. Nowhere was there a rent in the pigment-epithelium layer, which was separated from the layer of cones and rods by a stratum of the same finely granular mass. Each blood-vessel was surrounded by an aggregation of blood corpuscles which was very dense in the connective tissue of the adventitious coat, but became less so at a distance from the vessels. In certain places the

connective tissue between two blood-vessels was crammed with blood corpuscles. Those portions of the nerve-fibre layer, as well as those of the ganglionic and inner nuclear layers, which did not contain blood-vessels, were evenly permeated by isolated blood corpuscles. The tissue of the retina is nowhere destroyed by the accumulations of blood-cells. The membrana limitans interna in several places is raised by underlying aggregations of blood corpuscles, but no rent can be detected in it. No connection can be traced anywhere in the numerous preparations made between the collections of corpuscles in the outer coat of the blood-vessels and those contained within the walls of the vessels, and no rent in these walls could be discovered anywhere. The capillaries and veins were surrounded by wide, clear lymph-spaces, containing here and there a few isolated leucocytes. The walls of the vessels were corrugated and collapsed. Some of the veins were crammed with blood corpuscles. On transverse sections of the arteries they were found not to be completely surrounded by lymph-spaces. In some places a row of blood corpuscles was seen to traverse a lymph-space in an oblique direction from the wall of the vessel to the patch in the adventitia. Some isolated leucocytes were seen situated within the walls of vessels.

There were no changes whatsoever in the peripheric portions of the retina, and in the choroid.

In this case, as well as in those that have been heretofore examined microscopically, the extravasations had not taken place in consequence of rupture of the blood-vessels, but by diapedesis. No connection could be traced anywhere between the collections of blood corpuscles in the adventitia and those contained within the walls of the vessels. The retinal tissue occupied by the accumulation of these corpuscles was not the seat of destruction, as it would have been if a large mass of blood corpuscles had suddenly escaped. The even distribution of the blood-corpuscles throughout the layers of nerve-fibres and ganglion cells suggested the fact that the blood corpuscles had singly passed through the walls of the vessels. They collected in large numbers underneath the membrana limitans interna, where the resistance was lessened.

It seems a probable supposition, that the altered condition of the blood, combined with the enfeebled heart's action, caused an effusion of serum to take place into the tissue of the retina, as well as into the pericardium and the pleural cavities. The œdematous tissue compressed the veins and caused stasis in these vessels and the capillaries in consequence of which blood corpuscles were pressed through the distended walls of the vessels. The stasis and œdema were most marked in the vicinity of the optic-nerve entrance, especially in the direction toward the macula lutea. There the whole of the retina was thickened, and the nerve-fibre bundles were crowded asunder. The point of bifurcation of the central vein was marked by two dots, which could be made out macroscopically. The capillaries and veins were considerably distended, and some were crammed with blood corpuscles. The layers lying behind some distended vein were made to protrude toward the choroid. In some places a rupture had taken place through these layers, and the rent thus formed was filled with exuded blood-corpuscles, which detached the pigment-epithelium from the layer of rods and cones. In the region of the macula lutea, the pressure of the large accumulation of blood corpuscles underneath the membrana limitans interna prevented swelling of the retinal tissue. The accumulation was encircled by a fold of œdematous tissue consisting of the layers of nerve-fibres and ganglionic cells. The wide lymph-spaces surrounding the vessels were found to be empty, or contained only a few leucocytes. This fact can be explained by the theory, that the vessels being distended through life, had obliterated the lymph-spaces, so that the exuding blood-corpuscles passed directly into the loose connective tissue of the adventitia, which was found to be crowded with them. The number of these corpuscles was greatest in the immediate neighborhood of the blood-vessels, whilst, beyond the adventitia, only a few isolated cells could be found in the layers of nerve-fibres and ganglionic cells. This arrangement explained the ophthalmoscopic picture of the hemorrhages, which appeared in the form of bright-red dots and streaks closely following

the course of the vessels. They showed a distinct outline on the side nearest the vessels, whilst, on the other side, they had no distinct contours. In some places they formed complete sheaths surrounding the vessels. The blood-corpuscles which had penetrated into the layers of nerve-fibres and ganglionic cells showed a tendency to progress toward the macula lutea, and there to accumulate underneath the membrana limitans interna. The cause of this phenomenon seems to have been the fact that the pressure in the vitreous body was less than that in the œdematous retina. The collection of blood-corpuscles in this region could be seen with the ophthalmoscope as a large, dark-red, well-defined patch of oval shape, which was surrounded by several smaller patches. The extravasations caused the patient to see all objects tinged red.

The autopsy having revealed miliary tuberculosis of the lungs, I became inclined to consider the white dots, discovered ophthalmoscopically, as miliary tubercles of the choroid, though they were rather too bright for such. The microscopic examination proved that they were not tubercles, but only collections of exuded material in the neighborhood of enlarged veins containing a few blood corpuscles. These collections had burst through the retinal strata lying behind the veins and detached the layer of rods and cones from the pigment-epithelium.

THE PATHOLOGICAL ANATOMY OF PRIMARY SARCOMA OF THE IRIS.

By Dr. J. THALBERG, of St. Petersburg.

Translated by Dr. R. O. Born, New York.

(*Plate* i., *vol.* xiii.)

CONSIDERING the scarcity of primary sarcomatous tumors of the iris, the following case will offer an unusual interest, since it shows as the starting-point of the neoplasm the endothelium of the iris, and, in some places, its beginning encroachment upon the anterior surface and upon the cells of the reticulated layer of the iris (J. Michel).[1] The tumor had developed under the symptoms of an iridocyclitis, with severe remitting ciliary neuralgia, which necessitated the removal of the eyeball. Dr. Lange, who had enucleated the eye at the St. Petersburg Ophthalmic Hospital, was kind enough to give me the eye for further examination. I should like, however, to give a short clinical history of the case before I shall relate the results of the microscopical examination.

Olympiada J., sixty-four years old, was in March and April, 1880, operated on for cataract in both eyes; the operations were performed at the medico-chirurgical clinic, first upon the right and then upon the left eye. The left eye healed without reaction, but upon the right side she complained, since the operation, of a constant pain in the temporal region, which did not yield to any remedy, and which induced her to apply at the Ophthalmic Hospital. She was admitted July 1st, with the symptoms

[1] *A. v. Graefe's Arch. f. Ophth.*, vol. xxvii., 2, 1881, p. 171.

of iridocyclitis. In the left eye, some remnants of cataract; in the right, exudation in the pupillary space, with small remnants of cataract, deep episcleral injection, severe ciliary neuralgia, tenderness to pressure. The pain and injection are diminished by atropine, artificial leech, hydrate of chloral, blue ointment applied to the forehead, laxatives, etc.; the pupil is dilated, and on July 28th the patient is discharged. Sept. 8th, she returns with marked episcleral injection, ciliary neuralgia, tenderness to the touch, swelling of the iris, and turbid aqueous humor. Under the same treatment the symptoms disappear again, leaving only some photophobia, and on Oct. 21st she is again discharged. But since then the symptoms of irritation have from time to time reappeared, especially the severe ciliary neuralgia, and on Jan. 15th she is again admitted to the hospital. On Feb. 1st, after the eye had become quiet again, the secondary cataract is divided without cutting the iris. Morphium and hydrate of chloral are used to relieve the pain in the ciliary region. The injection and lachrymation are at times more, at times less, marked, but never disappear entirely. Pilocarpine and inunctions are not well borne, and are without effect. The patient is discharged on April 2d. At home the pain again increases, S = 0, and the patient herself asks to have the eye removed, which is done on April 17th by Dr. Weyert under chloroform narcosis. Healing normal. The left eye had, when patient was discharged, with +10D S = $\frac{6}{24}$.

The eyeball has been hardened in Müller's fluid, and is divided by a sagittal section. Only the inner half is used for examination. The sections are stained with chloride of gold.

The anterior chamber is filled with a soft, white mass which is firmly adherent to the iris. The iris and ciliary body are thickened, the lens reduced to a thin membrane except a zone 1 *mm.* broad at the periphery, which is swollen. The cornea also is thickened, the other parts of the globe show no macroscopic changes.

MICROSCOPIC EXAMINATION.—The white mass which fills the anterior chamber, appears under the microscope as a neoplasm made up of cells of varying size and shape; it begins at the pupillary margin, reaches beyond the sphincter of the iris, and in the thickest portion touches Descemet's

membrane. The pupillary portion of the iris with the sphincter muscle, is folded back upon the lens. It appears that when the pupil had been dilated, the pupillary margin had become attached to the lens capsule, and that this condition had remained when the pupil became contracted, thus producing the folding up of the iris. At the place where the iris is folded, the thickest and oldest portion of the tumor has been torn; the two portions have been detached from the iris, and the smaller portion grows upon the anterior lens capsule, the larger upon the cornea, leaving a free space between them (fig. 1 *s*). In this free space several layers of cellular elements have remained upon the fold of the iris, and from there a few projections have developed. From the smaller portion of the tumor cells extend around the shrunken lens, and, between posterior lens capsule and vitreous, reach the swollen marginal zone of the lens. These cells appear to enter through the opening which had been made by the secondary division of the lens capsule (fig. 1 *f*).

The cellular elements vary in size and shape according to their location, and to the rapidity of their development. Upon the fold of the iris and upon the opposite surfaces of the detached portions of the tumor are several layers of mostly flat cells with finely granulated protoplasm and 1–2 large nuclei (fig. 1 *s*).

The cells bear a great resemblance to the pavement epithelium of the cornea. Surface preparations show a fine mosaic of polygonal cells, in transverse sections they are short spindles with oval nuclei (fig. 4 *a*, *b*). By division of the nuclei of the large polygonal cells small cells have been formed, which at first have the same shape, but gradually pass over into round cells resembling in size and appearance the cells of the middle layer of the corneal epithelium (fig. 4 *d*). The tumor upon the lens capsule, the outer layer of the projections from the fold of the iris, and the inner third of the tumor upon the cornea are almost entirely made up from these round cells. By further division, round cells the size of lymph corpuscles are formed (fig. 3 *b*). The cells which extend around the shrunken lens are mostly spindle-

shaped, with two shorter or longer processes (fig. 4 *c*). Numerous cells of the same kind are found among the round cells upon the lens, but very few upon the corneal portion of the tumor. The central portion of the latter consists of small spindle cells with oval nuclei (fig. 3 *a*). The transition between the small round and spindle cells and the large round and polygonal cells, is formed by deeply stained round cells the size of lymph corpuscles, with a round nucleus, covered by a thin layer of protoplasm. The outer portion of the corneal tumor consists of round cells and nuclei of the same size but less deeply stained (fig. 3 *b*). The round cells are imbedded in a finely granulated, the spindle-shaped cells in a fibrillated, substance, which disappears in proportion to the increase of the division of the cells. In some places where the cells are very densely aggregated, their contours can sometimes not be distinguished any more; we see only round and oval nuclei imbedded in a uniform finely granulated substance (fig. 5).

The development of the neoplasm from the endothelium of the iris can be distinctly traced at the place where the corneal tumor passes over upon the anterior surface of the iris (fig. 2). The endothelial cells are enlarged; they appear upon transverse sections oval or spindle-shaped, but do not cover the iris as a continuous layer. At the place where the corneal tumor joins the iris, we find by division of the nuclei round cells with large nuclei surrounded by a small layer of protoplasm, at first forming a single layer upon the iris, but soon increasing to two, three, and more layers (fig. 2 *e*). Near the iris and cornea they retain this shape, but in the centre of the neoplasm they become spindle-shaped with long oval nuclei (fig. 3 *a*). The transition into the large polygonal cells at the fold of the iris is, as before mentioned, rather sudden, a small layer only of large and more deeply stained round cells intervening.

With the exception of a few small spaces near the fold of the iris, the neoplasm does not yet enter the iris. Even in the oldest portion, between the pupillary margin and the fold, where the iris tissue shows the greatest changes, the darkly pigmented iris is everywhere sharply defined from

the non-pigmented tumor. The latter can also, in making the section, be easily detached as a continuous layer of cells from the reticulated layer of the iris. In those places where the iris is affected, the adjoining cells of the reticulated layer produce by division similar round cells as are found in the neoplasm. Since, however, these cells are partly pigmented, we find also in this portion of the growth, besides the unpigmented cellular elements, some pigmented round and spindle-shaped cells and clusters of pigment, and the dark line between iris and tumor, formed by the outer pigmented layer of the iris, is interrupted. Where the clusters of pigment are interspersed between spindle-shaped cells, they have assumed an oval shape, as if they had been subjected to pressure.

The whole uveal tract shows an abundance of pigmented, round, oval, spindle- and star-shaped cells. Transverse sections of the blood-vessels of the iris show thick walls surrounded by a circle of round and oval pigmented cells (fig. 1 *g*.), and also the bundles of the ciliary muscle are surrounded with dark cells.

The ciliary portion of the iris, which is not yet covered with the neoplasm, is increased to twice its normal thickness by lymphod cells, some of which at least originate in the stroma cells of the iris. Toward the fold, and especially at the fold itself, the cellular infiltration increases considerably, and the iris is from three to five times thicker. The infiltration of cells is always densest near the endothelium and decreases toward the pigment layer. The pupillary portion of the iris is densely infiltrated with pigmented cells, large, irregular clusters of pigment and free pigment granules, between which only small tracts of round cells and nuclei are found. The pigmented cells are densest and the clusters of pigment largest in the outer layers; toward the sphincter the unpigmented round cells are numerous. We find in this portion of the iris all the shades from a light brown to a deep black. The clusters of pigment have an irregularly round or oval shape, without distinct nuclei. Numerous thick-walled blood-vessels have been formed in this portion of the iris. The sphincter has remained un-

changed. Also the pigment layer shows no change, only at the fold it appears thickened and of darker color.

The ciliary body is also thickened, but less than the iris, by an infiltration of cells and nuclei, which are more densely accumulated between the pigment epithelium and the ciliary muscle. There are many round cells at the insertion of the ciliary muscle into the sclerotic and in the inner layers of the sclerotic. From there cells have entered the anterior chamber and lie in clusters upon the iris. The choroid, retina, and vitreous show no changes.

At the equator of the lens are swollen fibres which are partly broken down into round, swollen masses resembling amylum granules. From the place where the pupillary margin is attached to the anterior capsule, toward the centre, the fibres disappear suddenly, the lens is thinner, and the anterior and posterior capsules are separated only by a thin fibrillated layer (fig. 1). The capsule of the shrunken portion is contracted into folds, and the sinuses of the anterior as well as the posterior capsule are filled with mostly spindle-shaped cells of the neoplasm and a finely-granulated substance. The epithelium of the anterior capsule is unchanged.

The cornea is thickened and pervaded by blood-vessels. In the corneal spaces we find sometimes clusters of round cells in place of the corneal corpuscles. Where the tumor touches the cornea, the accumulation of round cells has reduced the basis-substance to thin lamellæ. Descemet's membrane lies like a thin, undulated ribbon between the cells in the cornea and the adjoining tumor. The endothelium has been destroyed at this place. Blood-vessels traverse the cornea and enter the growth.

The endothelial cells of Descemet's membrane are swollen and become detached in shreds in making the sections. In some places, by division of nuclei, one or more layers of cells resembling pus corpuscles have been formed which either still adhere to Descemet's membrane, or lie in small clusters upon the iris and the neoplasm. By their 3–4 nuclei and broad granulated protoplasmic substance they are distinguished from the round cells of the tumor of equal

size, which mostly have only one nucleus and but little protoplasm.

A review of these notes shows us the development of a sarcomatous neoplasm of the iris in an old lady, apparently as the result of a cataract-extraction, developed from the endothelium and accompanied with severe remitting ciliary neuralgia. From its origin in the endothelium, from the form and size of the cells, we must interpret the growth as a sarcoma; and from the prevalence of the round cellular elements, it is a round-celled sarcoma. In its peripheric, most recently formed, portion, where, by division of the endothelial cells of the iris, round cells resembling lymph corpuscles have been formed, which, in the central layers, apparently by the pressure of the growing tumor, have assumed spindle shape with oval nuclei, the tumor has the appearance of a simple so-called granuloma. In the central, oldest, portion, however, we find a rather sudden transition from these small cells into large polygonal and round, distinct sarcoma cells, among which we find even some large spindle cells. By division of the large polygonal cells smaller ones are formed which at first retain their polygonal form, but by further division pass over into round cells the size of lymph corpuscles.

The neoplasm covers only the pupillary portion of the iris. The changes of the form and size of the sarcoma cells, and their rapid growth, seem to have caused the rupture of the tumor at the fold of the iris. The iris, where it is covered by the neoplasm, is greatly thickened by numerous round cells and nuclei, loose pigment, pigment cells, and thick-walled blood-vessels. The infiltration of cells is densest in the pupillary portion, and there more at the anterior surface of the iris than near the pigment-layer. The origin of the cells and nuclei can partly be traced to the stroma cells.

Where the tumor touches Descemet's membrane, the cornea is considerably thickened and infiltrated, and blood-vessels enter the tumor from the cornea. This is the only place where blood-vessels are seen to enter the growth. Although the tumor fills the anterior chamber and has

formed a vascular connection with the cornea, and although it passes through the opening in the centre of lens between the capsule and the vitreous, it has not entered the iris itself. The darkly pigmented iris and the unpigmented neoplasm are sharply defined, with the exception of a few small places in which the dark line is interrupted by the cells of the reticulated layer participating in the new growth. On account of the late and slow affection of the iris tissue itself it must be possible to successfully remove those primary sarcomata of the iris, as has, indeed, been demonstrated by Kipp [1] and Knapp.[2]

Explanation of Illustrations on Plate I., Vol. XIII.

FIG. 1.—HARTNACK, Ocul. 3, Obj. 7. (The other figures show the same magnifying power.) *C*, cornea ; *ee*, endothelium of the anterior chamber ; *i*, iris ; *p*, pigment layer ; *vk*, anterior lens-capsule ; *l*, lens ; *hk*, posterior lens-capsule ; *g*, blood-vessels ; *si*, sphincter of the iris ; *s*, sarcoma ; *f*, process of sarcoma encircling the lens.

FIG. 2.—Iris at the base of the tumor. *e*, endothelial cells ; *i*, reticulated layer of the iris.

FIG. 3, *a*, *b*.—Cells from the more recently formed portion of the tumor.

FIGS. 4 and 5.—Sarcoma-cells from the older portion of the tumor—*ad* from the surface, *b* in transverse section ; *c*, spindle cells.

[1] These ARCH., vol. v., p. 34.

[2] These ARCH., vol. iii., 2, p. 106, and vol. viii., p. 82,

THE COMPARATIVE FREQUENCY OF EYE DISEASES IN THE WHITE AND COLORED RACES IN THE UNITED STATES.[1]

By SWAN M. BURNETT, M.D.

OPHTHALMIC AND AURAL SURGEON TO THE GARFIELD MEMORIAL HOSPITAL AND TO THE CENTRAL DISPENSARY, WASHINGTON, D. C.

IN August, 1619, a Dutch vessel, with twenty African slaves on board, sailed up the James River in Virginia, and discharged its cargo at Jamestown. This was the first introduction of the negro savage among civilized white men in America, and from this ship-load and the others which followed, up to the time when the slave-trade in America was abolished, sprang with few exceptions all of that race at present to be found in Virginia, Maryland, and the District of Columbia. This is the most important instance in modern times where human beings in a state of savagery have been brought suddenly and on an extensive scale into the environment of the highest civilization and culture. The influence exerted by this civilization on their physical, mental, and moral condition is of the highest ethnographic interest, and has been studied, among others, by Nott and Gliddon.[2] But so far as my knowledge of American medical literature extends, no statistics have been published showing the comparative frequency of various diseases in these two races, so widely separated in the scale of civilization, when placed as

[1] Read, in abstract, before the Anthropological Society, Washington, May 20, 1884.

[2] "Types of Mankind," by J. C. Nott and G. R. Gliddon, Philadelphia, 1854.

nearly as possible in the same surroundings. It is a matter of scientific and medical interest to know how far this change of condition has influenced their susceptibility to an immunity from certain diseases; and their study may tend to show us how far certain doubtful diseases are climatic, zymotic, or constitutional. It has long been believed that the negro is prone to certain diseases, and to a greater or less extent free from others. He is but little affected, for example, with malignant paludial fevers, and is more liable to strumous and tuberculous affections, and scarlet-fever is much less fatal among the negro than the white;[1] but of detailed statistics on most of these points, I believe there are none.

That this difference in the susceptibility to disease should extend to the eye would seem highly probable, reasoning *a priori* on the evolution hypothesis, but no investigation in America has up to this time furnished any statistical evidence of the fact.

When I was placed in charge of the Eye and Ear Department of the Central Dispensary, I began to keep my records with a view of accumulating material for such statistics, each patient being entered as "white" or "colored." When there was any admixture of negro blood, even to a small extent, the patient was registered as "colored." This, it must be confessed, is hardly fair to the colored race, but under the circumstances it seemed unavoidable.

For the formation of the following table I have taken the three last published reports of my service at that institution. I have not drawn from any other source, because I wished to take as a basis of comparison those cases where the surroundings and modes of life were as nearly as possible the same. It will be observed that the numbers are small in comparison with those furnished by some of our great ophthalmic institutions, but they have the advantage of all having been examined by the same observer, and with great care, in view to this statistical presentation. I may be allowed to observe at the outset that it is hardly hoped that these

[1] In looking over the report of the Health Officer of the District of Columbia for 1883, I find that during the last five years the deaths from scarlet-fever per thousand of population have been for whites, 499, for the negroes, 139.

statistics will definitely settle any point. They are offered more with the design of inducing the publication of other observations in the same field of inquiry, not only in the United States, but also in other countries where the African race is to be found.

The total number of cases recorded was 2,325, of which 1,514 were colored, and 811 white (roughly ONE THIRD white and TWO THIRDS colored), and they have been classified according to the diseases as follows:

Diseases of the Conjunctiva.

	White	Colored
Conjunctivitis, catarrhal	149	304
" purulent	8	22
" scrofulosa	17	83
Ophthalmia neonatorum	4	5
Hyperæmia of the conjunctiva	8	9
Trachoma	44	
Pterygium	9	7
Ecchymoses of conjunctiva	3	7
Tumors	2	5
Episcleritis	5	15
Chemosis of conjunctiva		2
Circumcorneal hypertrophy of conjunctiva	3	5
Tumor of curuncle	1	3
Foreign body in conjunctiva	1	2
Pinguecula	1	6
Wounds and injuries		1
Varix of cul-de-sac	1	
	256	476

Diseases of the Cornea.

	White	Colored
Keratitis	50	228
" scrofulosa	14	48
Leucoma corneæ	28	83
" adherens	4	14
Foreign bodies in cornea	12	10
Ulcer of cornea	16	39
Staphyloma	2	9
Kerato-iritis	2	8
Abscess of cornea	4	12
Deposit of lead in cornea	1	
Keratoconus		1
Trachomatous pannus	2	
Tumor of corneal margin		1
Traumatic rupture of cornea	6	4

Keratitis bullosa		1
Burn of, from lime		1
Sloughing of cornea		1
	141	460

Diseases of the Iris, Choroid, and Vitreous.

	White	Colored
Iritis	17	75
Choroiditis	11	12
Closure of pupil	3	6
Sclerotico-choroiditis	1	3
Albinism	1	
Cyclitis	5	4
Opacities of vitreous	1	2
Prolapse of iris	2	6
Hemorrhage into vitreous	1	
Mydriasis		6
Iritis, traumatic		1
Sarcoma of choroid		1
Tubercules in ciliary body and iris		1
Rupture of choroid		1
Cyst of the iris	1	
Tubercular tumor of the choroid		1
Muscæ volitantes	1	3
	44	122

Diseases of the Lachrymal Apparatus.

	White	Colored
Dacryocystitis	13	28
Eversion of puncta	3	1
Stricture of nasal duct	12	15
Tumor of lachrymal gland		1
	28	45

Diseases of the Lens.

	White	Colored
Cataract, senile	38	68
" traumatic		6
Dislocation of lens		2
Nuclear cataract	1	
	39	76

Diseases of the Lids.

	White	Colored
Blepharitis	84	83
Entropion	11	1
Trichiasis	3	
Distichiasis		2
Abscess of lid	2	3

Emphysema of lid		1
Ptosis	1	3
Wounds of the lid	2	1
Chalazion	16	63
Ectropion	1	5
Hordeolum	3	7
Tarsitis	2	
Blepharospasm	1	
Tumors	4	5
Lupus of the lids	1	
Ankyloblepharon		1
Œdema of lids	1	4
Herpes of lids	1	1
Ulcer of lids	2	
Eczema of lids	1	2
Epithelioma of lids	1	
Symblepharon		2
	137	184

Diseases of the Muscles.

	White	Colored
Paralysis of external rectus	3	5
" " 3d pair	2	
" " sup. oblique		1
" " all the muscles	1	
Strabismus convergens	36	3
" divergens	1	2
Nystagmus	2	
Paralysis of orbicularis		1
	45	12

Diseases of the Globe and Orbit.

	White	Colored
Phthiss bulbi	5	13
Hydrophthalmus		1
Panophthalmitis	3	9
Symp. ophthalmia	3	3
Exophthalmus	2	1
Periostitis of the orbit		1
Tumor of the brow	1	1
" " " orbit		1
Supra-orbital neuralgia	3	3
Abscess of the orbit	1	1
Gun-shot wound of the orbit		1
Orbital cellulitis		1
Enlargement of the frontal sinus	1	
Foreign body in eye	2	
Wound of globe		1
	21	37

Diseases of the Optic Nerve and Retina, and Glaucoma.

	White	Colored
Atrophy of optic nerve	14	13
Retinitis albuminurica	2	5
Amaurosis	1	7
Retinitis sympathetica		1
" specifica	3	3
" pigmentosa	1	2
Neuro-retinitis		3
Myxoma of optic nerve	1	
Glaucoma simple	4	14
" inflam.	1	2
" secondary		1
Hyperæmia of retina	2	2
Glioma of retina		1
Retina, detachment of	2	
	31	54

Anomalies of Refraction and Accommodation and Vision.

	White	Colored
Asthenopia	5	7
Hypermetropia	27	19
Astigmatism	7	4
Hemianopsia		1
Hemeralopia	1	4
Paralysis of accommodation	1	
Myopia	18	6
Presbyopia	6	4
Diplopia	1	2
Color-blindness	1	
Mydriasis	1	
Nyctalopia	1	1
	69	48

COMMENTS.

Taking CONJUNCTIVAL DISEASES as a whole, they are of about the same relative frequency in the two races. Of *scrofulous conjunctivitis* (which includes herpes of the conjunctiva of the sclera and cornea) the proportion is very much larger in the colored race. In fact all the forms of so-called "*strumous ophthalmia*" are relatively more common among the negroes. *Pterygium* does not seem to be so common. The slight difference in the number of cases of *ophthalmia neonatorum*, *episcleritis*, and *circumcorneal hypertrophy of the conjunctiva* can be looked upon as purely accidental.

When, however, we come to *trachoma* we are confronted with the remarkable fact that whereas the whites had forty-four cases, the negroes *had none*, though their proportional share would have been at least eighty. This is too great a discrepancy to be accounted for by a mere coincidence. In 1876, in a paper read before the Internat. Oph. Congress in N. Y., on "Trachoma as Influenced by Race," I called attention to the fact that, according to my observation, the negro seemed to enjoy an immunity from this disease.

The accuracy of this observation was called in question by Dr. H. D. Noyes, who claimed that they had "colored trachoma" in N. Y. I was not prepared on that occasion to support my position by any statistics, and these that I now offer are the first that I have been able to collect since that time, and to my mind they are conclusive. I would not go so far as to say that the negro *never* has trachoma; I can only say that I have never seen a genuine case of that disease in that race—even among the mulattoes. They have papillary conjunctivitis, which is often classed under the general term "granular lids," but clinically that is a very different disease from the one which is known and recognized under the name of trachoma. About the histological peculiarities of trachoma; in what it consists, and of its exciting cause, there are various opinions. We have not time here to discuss the question as to whether the trachoma follicle is produced by inflammatory alterations in the mucous membrane, or whether it is a special formation,[1] or whether it is but a modified form of purulent conjunctivitis, as is held by Arlt.[2]

The *clinical* features of genuine trachoma are unmistakable. It is an affection of the conjunctiva, usually chronic in its character, attended with remissions and exacerbations, extending most frequently through a number of years, and resulting, with very few exceptions, whether left to itself or under any form of treatment, in a greater or less loss of substance of the conjunctiva, which is replaced by cicatricial

[1] See exhaustive papers by Mandelstamm in *Grafe's Archiv*, xxix., 1, and by Raehlmann, *ib.*, xxix., 2, in which the different views from a histological standpoint are brought down to date.

[2] *Klin. Darstill. d. Krankheit d. Auges.*, '81.

tissue. Such affection of the conjunctiva, which forms from four to six per cent. of all the cases presenting themselves for treatment at the eye clinics of the principal Eastern cities of the United States, I have not seen—in a single instance—in the negro.

If it should turn out that the negro does not suffer from trachoma, even when the surrounding conditions are such as usually develop it in other races, we have a new element that must be taken into account in studying that most important eye-affection. We can then no longer regard it as a mere local disease, to be treated by local remedies alone. It must be considered as the expression of a dyscrasia. In their anxiety to find a local specific for trachoma, investigators seem to have forgotten the possibility of a general cause lying back of it—though there are many facts which point strongly to this latter fact. It has been looked upon as a contagious disease, because it often affects a large number of the inmates of asylums and other public institutions at the same time. This fact would indicate a general operating cause, quite as well as a contagion, as an origin for the epidemic. Then again, the natural history and course of the disease have not been sufficiently studied, and we can not be sure that it is not, in a degree, self-limited. The large number of local applications which have from time to time been recommended as specifics for trachoma—embracing every known astringent in the materia medica—is *prima-facie* evidence that there is no one remedy in which the whole profession has confidence. Attacks of trachomatous inflammation subside under all kinds of treatment, caustic, emollient, mildly and strongly astringent; and there are many cases, as all of us know to our chagrin, which have not improved, and which even got worse, under any kind of treatment, which have gone on to as favorable termination, without local interference, as have the cases where we have used our favorite remedy. That the profession is still looking for a reliable remedy for trachoma is sufficiently evidenced by the great interest felt in the results of the jequirity treatment. The highly interesting and instructive symposium in the last number of these ARCHIVES shows this,

but shows also that even jequirity leaves much to be desired. In fact, from a careful reading of these views, the impression is strong in my mind that jequirity is not so much a remedy *for trachoma*, as for the *results* of trachoma.

If trachoma *is* a mere local disease there can be no reason why the negroes who have the same environment as the Irish, for example, in regard to habitations, food, light, etc. should not have it in the same degree. When I was a boy, a railway was built through East Tennessee, in the construction of which both the Irish and negroes were employed. My father, who was a physician and practised among them, told me that chronic sore eyes were very common among the Irish (and we all know what that was), whereas such affections of the eyes were not met with among the negro laborers. And it was there that, at the beginning of my professional career, my attention was first directed to the absence of trachoma in the negro, though it was very common among the whites.

For a number of years the thought has been growing upon me that the trachoma-granule, follicle, or whatever name we may choose to call it by, is a deposit of material which is analogous in its character to tubercle. At least they both seem to have this in common—that when they disapper the tissue in which they are imbedded is destroyed, and a cicatrix is the result.[1] There is another similarity also in the successive crops of these deposits which may occur. I am inclined to regard each fresh attack of trachomatous inflammation as the manifestation of a fresh deposit, and where we have a series of them, as we often do, through a number of years with these resultant cicatrices, we have that total destruction of the mucous membrane so commonly met with in the ancient cases. No other inflammation of the conjunctiva shows any such destructive tendency except the diphtheritic.

In the treatment of trachoma, hitherto our attention has been principally directed to the inflammatory symptoms which accompany it, to the *conjunctivitis*, but should the views now advanced, and seemingly supported by an array

[1] Similar views are held, I believe, by the younger Sichel.

of facts, be found correct, we should begin the study of trachoma from a new standpoint, and our therapeutic agencies will have to be directed to the prevention of the trachomatous deposit, and the general condition of the patient will receive first attention.

Since our failure to cope with it as a local disease has been so lamentable, we might try what our success will be when it is treated as a dyscrasic affection. Local applications, however, would even under these circumstances not be neglected in combating the inflammatory complications.

Of the *tumors* of the conjunctiva, one case of *lipoma*, from the comparative rarity of its occurrence, may be worthy of being placed on record.

It was on the right eye of a colored girl, twenty years of age. She said she had had a small lump at the outer corner of this eye since birth. It had remained about the size of a pea until the last year or two, when it began to grow rather rapidly. It extended forward in the palpebral space to within six millimetres of the corneal margin, and backward beyond the equator, and was freely movable on the sclera, and the conjunctiva was slightly movable over it, except at the centre, where it was fixed. There was no pain or inflammation, but latterly the movements of the ball outward were somewhat impeded, and caused a feeling of discomfort. An incision was made in the conjunctiva over it, and where it was adherent dissected off. The tumor was then enucleated without difficulty. The wound healed kindly, and only a small scar remained. The tumor measured $2\frac{1}{2}$ *cm.* in length, by $1\frac{1}{2}$ in breadth, and was $\frac{3}{4}$ *cm.* thick.

A histological examination showed it to be purely lipomatous in its structure.

Among diseases of the CORNEA, we find in the negro a disproportionately large number of inflammations of all kinds, as we should expect would be the case in a race so decidedly "strumous," and *parenchymatous keratitis*, with Hutchinson's teeth, is met with, though not more frequently than among the whites.

Of IRITIS the percentage among the negroes is twice that among the whites. The large amount of pigment in the uveal tract would cause us to suspect some modification of

inflammation in that tissue, and our tables show an increased liability to inflammation on the part of the iris. On the other hand, *choroiditis* and *cyclitis* do not appear to be relatively so frequent. The percentage of *syphilitic iritis* in the negroes was not greater, as far as could be determined, than among the whites. As regards the prognosis of inflammation of the uveal tract in negroes, it is very difficult in a dispensary service to form reliable opinions; but I do not think it is so unfavorable as Dr. C. S. Bull seems to believe it is. Certainly many cases yield as promptly to treatment and make as favorable recoveries as any cases among the white population.

I saw one case of what I took to be *tubercle* of the iris and ciliary body. Unfortunately the patient passed from observation before a positive diagnosis could be made.

It was in the left eye of a mulatto girl, seventeen years of age. She had noticed trouble in that eye for some weeks before I saw her. There was a sharply circumscribed tumor—nodular, and with fine vessels running over its surface—at the base of the upper part of the iris, measuring $2\frac{1}{2}$ *mm.* in width and 3 *mm.* in length from above downward, and reaching nearly to the posterior corneal surface. There was much ciliary injection and pain. In a week from the time I first saw her, a swelling of the adjoining sclera was observed, which was gradually increasing up to the time she disappeared from observation. As she showed other symptoms of tuberculosis, I was inclined to regard this tumor of that nature.

The case of a *large tubercular tumor of the choroid,* occurring in a negro girl, I have reported in full in vol. xii., No. 4, of these ARCHIVES.

Of *sarcomatous tumors* of the choroid, I have had one in the negro man, which in its history and course did not differ in any important particulars from the cases usually met with. The patient was lost sight of after the enucleation, and I have no knowledge as to whether it returned or not. It was a pigmented sarcoma, though not more so than I have seen in the white race, and was confined to the anterior portion of the choroid.

Diseases of the LACHRYMAL apparatus appear of about the same frequency in the two races. The same may be said of CATARACT.

While the *paralyses of the* OCULAR MUSCLES are met with nearly equally in whites and negroes, *strabismus convergens* is comparatively rare in the negro. There were thirty-six "white" cases, and only three "colored." Refractive troubles being, as we shall see, less common among the colored race, this fact seems to support the theory of the dependence of strabismus on errors in refraction.

In LID AFFECTIONS a relative excess of *blepharitis marginalis* is noted among the whites, while *chalazia* are about twice as frequent in the negro. I would observe in passing that the orbicularis muscle is very strongly developed in some of this race, a fact to be taken account of in operations without an anæsthetic.

SYMPATHETIC OPHTHALMIA probably affects the two races equally.

The negroes are not more liable to *retinitis pigmentosa* than the whites. They are subject to *neuro-retinitis* and to *retinitis albuminurica*. There was one case of *glioma of the retina*, in a negro girl four years old. When first seen, the tumor had already burst through the walls of the globe. The optic nerve was not affected beyond the lamina cribrosa, and there was no return of the growth five months after the operation, when death occurred from meningitis consequent upon mastoid disease due to chronic purulent inflammation of the middle ear.

GLAUCOMA, especially the *chronic simple form*, is very frequent among the negroes, more so, it would seem, than among the whites.

Of REFRACTIVE anomalies, much fewer cases were observed among the negroes than among the whites. We must, however, not conclude too hastily from this that they do not exist in the same proportion. I am inclined to think that hypermetropia is to be found to about equal extent in both races, but the asthenopia attendant upon this condition, and which brings them to examination, is not brought out in the negro to the same extent as in the whites of the

same social status. But it is certain that *myopia* has not yet got such a hold on the negro as upon the white race. What effect systematic education is going to have upon the development of this condition we are not yet in a position to state with any degree of positiveness. I am making what observations my opportunities allow on this point, and may be able to report upon it in the future. Only six cases of "colored" myopia were noted, whereas the whites had eighteen.

Several cases of *night-blindness* were observed. There was no exciting cause of a special nature discoverable. I was therefore compelled to fall back upon the supposition of some form of malnutrition as an origin for the condition. In three cases the collections of small air bladders on the conjunctiva, noticed by other observers in such cases, were present.

Operations.

	White	Colored
Extraction of cataract	11	23
Discision " "	3	4
Excision of chalazion	17	42
Enucleation of eyeball	13	14
Operations for entropion	5	
" on lachrymal apparatus	24	14
Removal of foreign bodies in cornea	10	8
Iridectomy	15	18
Iritomy		2
Detachment of ant. synechia	1	1
Abscess of lids opened	5	3
Operation for strabismus convergens	27	2
" " " divergens	1	
Removal of conjunctival tumor		3
" " tumor from brow		2
" " " inner canthus	1	1
" " " lids	1	3
Division of outer canthus	2	
Optico-ciliary neurotomy		1
Removal of pterygium	3	4
Tattooing of cornea		5
Saemisch's operation		1
Excision of staphyloma	1	
Plastic operation for ectropion		1
Detachment of symblepharon		1
Sclerotomy		1

Operation for ptosis	2	
Removal of myxoma from o. n.	1	
Total	145	155

REMARKS.—Of the eleven *extractions* of *cataract* in the whites there was one failure, due to constant vomiting for forty-eight hours after the operation. Of the twenty-three in the negroes two eyes were lost through suppurative panophthalmitis. Both cases were in women. One died two weeks after the operation, and the other had been bed-ridden for four years. I should state that these operations were undertaken only at the earnest solicitation of the patients. I am satisfied from my experience that, other things being equal, the negro offers as good a general prognosis in eye-operations as the white race, though I am inclined to think they are rather more liable to reactions on the part of the iris after extractions.

SECONDARY SEPTIC OPHTHALMITIS.

By P. H. MULES, M.D., Manchester, England.

THE paper in your last issue by Professor Theobald, on the "Pathology of Sympathetic Inflammation of the Eye," will surely arrest the attention of ophthalmologists, not only as an attempted reposition of the views of Mackenzie and the older writers, but as a challenge to the Septicists. Certainly Professors Leber and Snellen require no assistance in fighting their battle of "septic invasion" against that of "reflex neurosis," but as a convert to their views I venture to formulate my own in this paper.

There appears still to be a fatal fascination in the term "sympathetic ophthalmia"; the word "sympathy" is so full of suggestion, and yet so vague, that whilst binding its disciples to nothing, it opens possibilities for wide application.

In the selection of the term "sympathetic ophthalmia," we have ample evidence from his writings that Mackenzie was guided solely by the clinical aspect of the disease, which, taken alone, unquestionably favors what we shall endeavor to prove to be an incorrect view of its pathology. Had he called the affection by any other name, we dare venture to say that the necessary reclassification of these diseases would not have been so long delayed.

We know that the name sympathetic ophthalmia is not used to denote any clearly defined disease of one portion of the eye, but is an expression of a complex series of pathological phenomena occurring in an otherwise healthy organ from *injury or disease of its fellow.* The words in italics appear to me fatal to the believers in "reflex neurosis."

There must be injury or disease of the one eye to initiate sympathetic inflammation of the other. Even operative interference, carried to a most successful issue, and in no way affecting the operated eye prejudicially, will produce sympathetic inflammation in its fellow (cases 1 and 2), and this is quite what we might expect if we consider from how small a surface the whole body may become infected by micro-organisms—whilst reflex irritation of the severest type, prolonged for many months and causing total blindness for the same period, passes away without leaving a trace of the storm (case 3). The believers in "reflex neurosis" assume that injury or disease of the one eye causes sympathetic inflammation of the other by injury to the optic or ciliary nerves, or incarceration of the ciliary nerves alone, with subsequent structural change. If this is so,

---

Case 1.—From my own case-book. J. Hartley, a man, æt. sixty-four, attended my clinique suffering with mature senile cataract of the right eye, the left quite healthy.

Vision, tension, and field, normal. The opaque lens was removed satisfactorily, the clinical notes remarking on the absence of *any* complication. He left the hospital in two weeks. Vision = J. 1 and 6. 12. with appropriate glasses. Six months after the operation, a plastic irido-cyclitis with descemitis, typically sympathetic in appearance, commenced in the unoperated eye. It caused entire destruction of the globe, which was eventually removed, the right operated one retaining good vision.

Case 2.—Hirschberg (A. O., vol. viii., p. 224) records a similar instance of sympathetic inflammation occurring after *apparently* normal healing of the cataract-extraction.

Case 3.—Power (On the Relations between Dental Lesions and Diseases of the Eye, 1883, p. 30) refers to Galezowski's case of *amaurosis* in a man, æt. thirty, who suffered from a carious tooth, through which he had managed to introduce a foreign body into the antrum, and thus keep up continuous irritation, resulting in absolute blindness and periodic attacks of pain. Galezowski found the pupil fixed and insensitive to light. He determined on removal of the first molar, at the extremity of the fang of which he found a splinter of wood. Pain ceased on withdrawal, and within nine days vision was perfectly restored, after an absolute blindness of thirteen months.

surely wounds, or diseases of the orbit implicating these nerves, yet without direct injury to the one globe, should occasionally cause sympathetic disease of its fellow. Is this so? The only instance I have met with, or to which I can refer, is recorded below (case 4), but here again we have stronger evidence in favor of "secondary septic invasion" than of "reflex neurosis," because it was evident that the disease could not be propagated from the left to the right side before the interior of the left globe was in a state of preparedness, the irritation of the nerves outside the globe being evidently insufficient in support of this side (case 5).

If, then, the most likely causes for the production of sympathetic disease by a reflex neurosis fail in accomplishing such results, what shall we say in favor of a theory

---

CASE 4.—Henry Tucker, æt. thirty-six, was struck in the upper part of the left orbit, to the inner side of the lachrymal gland, the globe remaining uninjured; the foreign body was supposed to be a piece of hammer-head. The wound healed kindly, and for two years he suffered no inconvenience; at the expiration of that time he felt the left eye growing dim (*the eye of the injured side*). He applied to the hospital and to my clinique, where I found iritis, faint descemetitis progressiva, neuro-choroido-retinitis following one another in a steady sequence. After many months of treatment, during which the right eye passed through a similar storm, the disease slowly abated. The affection in both eyes having been in appearance typical of that called sympathetic ophthalmia. After five years, during a portion of which he required to be led about, the vision improved from shadows to J. 1 and 6. 12. and six months since he reappeared and pointed to a sharp point in the orbit—evidently a foreign body; it was removed with difficulty, requiring great force, and found to be a long piece of steel, $\frac{7}{8}''$ in length, projecting backward and downward into the orbit: he is now in full work.

CASE 5.—Contrast this with the case mentioned by Power (On Relations between Dental Lesions and Diseases of the Eye, 1883, p. 5), where, for twelve years, a man carried a large portion of metal imbedded in the optic nerve; and, although suffering from "SYMPATHETIC IRRITATION," removal of the globe and foreign body produced an *immediately* good result.

which seeks to pin to such Protean maladies as "reflex neuroses" an affection which, however complex its course, always has a certain definite personality which enables us to detect its onset.

The interest centres in the fact that the foreign body caused primary disease of the eye of the injured side—presumably septic. The septic invasion of the second eye took place in the usual manner, the disease remaining through its course and progressing to recovery in spite of the presence of the foreign body which originated it (the opposite of Galezowski's case of true "reflex neurosis,") showing that, in this case at least, there was no SYMPATHY.

Snellen well says: "To attribute sympathetic cyclitis to reflective nervous action makes of it an isolated fact"; and he goes further to say "that all proof of such is wanting." Can we then explain, by any theory or assemblage of facts, the reasons why injury or disease of the one eye should produce the pathological changes known as sympathetic inflammation? We think we can. The theory of a "reflex neurosis" from injury to, or change in, the optic or ciliary nerves, producing disastrous inflammatory change, had long been unsatisfactory to exact minds, and after numerous careful dissections by competent observers of eyes which had caused sympathetic disease, it was demonstrated that incarceration, swelling, or other change of the ciliary nerves was, in a large percentage of cases, not to be found; therefore, the cause could not be invariably attributed to direct irritation of these nerves. What constant change could be found, to which such affection might be due? During the investigation it was noted (where it was possible to demonstrate such appearances), that the perineural lymph-spaces —especially at the posterior part of the eye—were dilated. This led to a careful investigation of their contents, which were found to consist of moving micro-organisms, and lymphoid cells; and, further, that the uveal tissue was densely packed with similar bodies, such bodies being also found in the secondarily affected eye, and it only required the further evidence supplied by Max Knies (Bericht Ophth. Ges. zu Heidelberg, 1879, p. 52,) who traced these

micro-organisms up the perineural lymph-spaces of the one eye, and down those of the other, to substantiate the theory of Leber and Snellen.

Holding this view we have the keynote to the admitted difficulties of prognosis in injuries to the eye.

It is evident that here are two factors: the first, the development of micro-organisms; the second, the perineural lymph-space dilatation. It is possible for each to exist without the other, but on their consentaneousness depends the affection of the fellow-eye. And on this also depends the lapse of time, even amounting to years, that the affection occasionally takes to develop, and the uselessness of removing the primarily injured eye, solely on the plea of its having *originated* sympathetic inflammation. It may be taken for granted that it has done its worst as a *propagator of micro-organisms*, and its removal will in nowise abate the disease *per se;* but as an originator and promoter of sympathetic irritation, each case must be treated on its merits. In "*sympathetic irritation*" we have the true "reflex neurosis," but it is in nowise identical with sympathetic inflammation, the relation of the two being as the storm-warning to the storm,—it cannot originate it; but sympathetic inflammation being present, sympathetic irritation may retard, if not prevent, its recession.

This is, I think, the explanation of those cases of relief following enucleation of an injured eye, which are adduced as examples of the cure of sympathetic inflammation by enucleation. They are seen daily, in the immediate relief and improvement of vision, caused by the removal of a painful lost eye, where no suspicion of sympathetic inflammation exists. That there is a capability for a diseased or injured eye perhaps to originate, surely to keep up, certain forms of conjunctival or corneal irritation in its fellow which pass away on its removal, cannot be disproved, and they may well be due to "reflex neurosis"; but they have nothing in common with sympathetic inflammation, for the *one point* in these affections is an immediate abatement or cure on the removal of the offending stump, and I look forward to a careful re-classification of such diseases, where the

term sympathetic shall be confined to such as the above, and "*secondary septic ophthalmitis*" claim its full significance.

The occurrence of WHITE CILIÆ during the course of sympathetic inflammation has been adduced in favor of "reflex neurosis," but white ciliæ occur very rarely; and when they do so, the change is not restricted to sympathetic inflammation. I have attending my clinique at the present time two cases. The one, of sympathetic inflammation of the right eye following injury to the left, in a girl æt. fourteen. In her all the ciliæ of the right upper lid have become snow-white. The second is a young woman æt. twenty-eight, with injury to the right eye, implicating the iris and lens. In this case the left eye is perfectly healthy, but the right upper ciliæ are snow-white. Both blanchings of ciliæ occurred within eighteen months of the injury.

The CLINICAL FACTS adduced in favor of "reflex neurosis" are tenfold stronger in favor of "septic infiltration." In Galezowski's case the removal of the affected tooth and foreign body caused almost immediate resumption of sight.

In not a few cases of active sympathetic inflammation removal of the primarily injured organ is the signal for a fresh outburst, showing at least that if it does not increase the danger, it has no influence in abating it.

The invariable cyclitis in true sympathetic inflammation is explainable on the grounds of the importance of the long ciliary nerves, and the presumption that lymph-channel dilatation could, with certainty, be found around them; whilst the rarer forms of neuro-retinitis, with transient cyclitis, are explainable by the micro-organisms passing through the lymph-channels of the optic and short ciliary nerves in preference to the long ciliary nerves, and their finding sufficiently strong resistance to their reproduction and extension. Further, let us mark carefully the *appearance* of an eye during an outbreak of *malignant* "sympathetic inflammation": the rapid and profuse exudation—the frequently-recurring outbreaks, yet without periodicity—and the well recognized facts that no sympathetic inflammation

commences before three weeks have elapsed, whilst it is favored in its onset by heat and moisture,[1] and we have far stronger presumptive evidence for germ invasion than for neurotic influence. An objection has been raised, that if sympathetic inflammation was due to septic invasion the micro-organisms would not confine themselves to the organs of vision, but would extend beyond the limits we at present assign them. It is, I think, a fair assumption (based upon the fact that sympathetic inflammation is confined absolutely to the human species), that there is a special micro-organism whose purpose is fulfilled by the induction of *ocular* "sympathetic inflammation." The advice given by Dr. Alt, and quoted by Dr. Theobald, for the removal of a long portion of optic nerve for the prevention of sympathetic inflammation, is of practical value in preventing the migration of micro-organisms, for it is quite possible thus to divide beyond the dilated lymph-channels of both optic and ciliary nerves; and where, as in the case of Dr. Vosè Solomon, sympathetic inflammation occurred after the enucleation, the micro-organisms were in course of transit, and the removal of the infecting eye caused neither delay nor need it have hastened the result.

In conclusion I would add that the acceptance of this view of a secondary septic ophthalmitis, whilst increasing, if possible, the perplexities and gravity of the prognosis, establishes a rational starting-point for such treatment as can affect the disease and does not leave it to the haphazard empiricism which was formerly the fate of sympathetic ophthalmia.

May, 1884.

[1] Brailey: Trans. London Med. Congress, 1881, vol. iii., p. 38.

THE UNEQUAL CONTRACTION OF THE CILIARY MUSCLE.

By S. J. BUMSTEAD, M.D., of Decatur, Ill.

ALTHOUGH some mention of the possibility of the unequal contraction of the ciliary muscle, with its influence in causing apparent astigmatism, or concealing a real astigmatism, has been made during the past five years, and a few cases cited tending to establish it as a fact, it has seemed to me a doctrine not yet accepted by the majority of oculists. It may be that this conclusion is arrived at because of my lack of access to *all* the current ophthalmological literature. Some years ago and, I believe, before I had seen any suggestions of this kind, it would occasionally occur to me in determining the refractive condition of certain eyes, that an unequal contraction of this muscle must take place, in order to explain the phenomena intelligently. The following cases will illustrate this action.

Mrs. E., thirty-four years of age, in excellent health, occupation librarian, has complained for several years of asthenopic symptoms, and was conscious that vision for distance was slightly defective; but the difficulty experienced with her eyes in prolonged close work, was what led her to consult me. S in both eyes = $\frac{20}{30}$, and R E with —30 cy., axis horizontal, S = $\frac{20}{20}$; L E with —30 cy., axis 170°, S. = $\frac{20}{20}$. She had used + 60 and + 48 for reading, but found they did not relieve her asthenopia. Her range of accommodation with the above-mentioned concave cylinders appeared to be all that was desirable, in so far as a short trial could determine it. A solution of atropia, 1:125, was then

applied, and after complete paralysis of accommodation, a further examination was made. It was then found that the above concave cylinders were of no use, and contrary to the first determination the R E required + 36 cy., axis vertical, with which S again = $\frac{20}{20}$. The L E required — 48 cy., axis 170° (instead of — 30 as before), combined with + 30 cy., axis 80°, and with this S again = $\frac{20}{30}$.

The spasm of the ciliary muscle it will be noticed was greatest, or affected the lens the most, in the vertical meridian in the right eye, and in degree corresponding to the difference between a lens of 30- and one of 36-inch focal distance, viz.: $\frac{1}{180}$. Before the action of the mydriatic the V M required — 30, while after this acted, nothing; and this excessive convexity in the meridian seemed to be caused by the efforts of the ciliary muscle in bringing the hypermetropic H M up to normal = + $\frac{1}{36}$. With the L E before paralysis of accommodation the V M (nearly that, 80°) required — 30 cy., axis 170°, to bring it to the emmetropic standard, and the H M was brought up by the action of the muscle. After the mydriatic, the V M required only — 48 cy., a decrease in the convexity of $\frac{1}{30} - \frac{1}{48} = \frac{1}{80}$; while the H M required + 30 cy. to bring it to normal and = $\frac{1}{30}$; while the real difference in this eye of the action of the ciliary muscle upon the lens in the different meridians was $\frac{1}{30} - \frac{1}{80} = \frac{1}{48}$, a much greater inequality than in the other eye. This lady did not obtain the relief she undoubtedly could from the use of these glasses, only because she thought she could not submit to the loss of time necessary to break up with atropia the ciliary spasm, which returned after the first use of the mydriatic, and has not as yet been repeated, as advised by me.

The next case was as follows:

Miss E. L., age thirteen, was brought to me, on account of rapid deterioration of vision, Oct. 20, 1882. Though conscious of some difficulty with her eyes for several years, she had been able to continue her work in school until four weeks previous to her visit to me. R E, S = $\frac{20}{200}$; L E, = $\frac{20}{100}$. With — 14 R E had S = $\frac{20}{30}$ partly, and L E with — 15, same. When the L E had — 16 placed

before the V M only, S = $\frac{20}{20}$, and with — 24 before H M, S was also $\frac{20}{20}$. Here was a difference of $\frac{1}{16} - \frac{1}{24} = \frac{1}{48}$ greater power over the convexity of the vertical meridian of the lens, or lens and cornea together, than of the horizontal. This girl, after her eyes had been thoroughly paralysed by atropia, did not require any cylindrical lens, and much less concaves as most readers will surmise, but really needed + 18 for both eyes, which gave her S = $\frac{20}{20}$, and with these she has had perfect use of her eyes since.

It will be remembered that the first case also exhibited a greater convexity in the vertical meridian, amounting in one eye to $\frac{1}{180}$, in the other $\frac{1}{80}$, while in the only eye of the second case of which I had preserved the record, the difference was $\frac{1}{48}$. It is probable that extended experiments upon emmetropic eyes, as to the ability by contraction of this muscle to overcome concave cylindrical glasses, will determine upon which meridian the ciliary muscle exerts the greatest power. Inasmuch as the slight degrees of astigmatism of the regular kind, existing in so many, and not requiring correction, is nearly always in the vertical meridian as indicated by a shorter radius of curvature, we would expect it to be in this meridian. In order to obtain some information upon this point, I made some tests of this kind upon my own eyes, which are emmetropic, although Green's lines are much impaired in clearness of definition and equality by a + 144 cy., whose axis is vertical. My age is forty-three, and have for twenty-two years had to use + 40 and latterly + 30 glasses for close work, in consequence of diphtheria. I first used the stenopæic slit, one millimetre in width, and then cylindrical concave glasses. With the stenopæic slit there was but little difference in the apparent power exerted over the two principal meridians by this muscle, as with the slit placed in both positions concave glasses as high as — 15 could be overcome and S still = $\frac{20}{20}$, and the lines look equal, or very nearly so. The use of the slit is probably not so fair a test of this as the use of the cylinders, as the former acts in this case much as the pinhole in a card increases vision in high degrees of ametropia. Even with the slit, however, there was a little greater facility when placed in the horizontal meridian. When I resorted

to the use of the concave cylinders I found the best I could do was — 24 cy., axis horizontal, while with the axis vertical only — 36 and — 30 could be overcome. I find in these ARCHIVES, from and including vol. iv., to the present time, the following allusions to this subject:

In vol. viii., page 544, an article by Dr. H. Gradle in *American Journal Med. Sciences*, Jan., 1879, is referred to as a case given which seemed to show that cases of regular astigmatism may be partly or wholly corrected by unequal contractions of the ciliary muscle. In vol. xi., page 110, M. Landesberg, in *Graefe's Archives*, makes the observation that he has seen regular astigmatism in cases of myopia with or without spasm of accommodation, and hyperopia with spasm of accommodation as a result of this anomaly, and that said astigmatism disappeared completely upon proper treatment. In vol. xi., page 497, Marckwort speaks of an astigmatic accommodation of the lens being frequently found in young subjects, which disappears after the energetic use of mydriatics. In vol. xii., page 508, is a reference to an article by Nordenson, in which he concludes that the lens generally corrects the corneal astigmatism, and acts as a convex cylindrical glass whose curvature in its various axes varies in consequence of unequal contraction of the ciliary muscle.

In conclusion, I would refer to the remarks of Dr. Reynolds of Louisville, Ky., as given in vol. x. of these ARCHIVES, page 234, in reference to the correction of his own case of simple astigmatism. He claims that a proper correction of this by a convex cylinder does not give relief, but that a concave cylinder, somewhat weaker in negative refractive power than the convex cylinder necessary to correct, when placed with its axis at right angles to the axis of the hypermetropic meridian, does do so, and enables him to use the eyes with comfort. He is disposed to attribute this to an unusual power of the ciliary muscle, but if this power was equal, is it not clear that when the concave cylinder was placed before such an eye that an increased power over the lens must result in the meridian opposite to the hypermetropic one in order to maintain vision at the normal? And if so, there must exist in such eyes great inequalities in the power of this muscle over the lens at one

and the same time, and which at once suggests its proper dissipation and relief without possible injury by the prolonged action of a mydriatic. Burnett has also spoken of this method of relieving certain cases of h. astig. as quite successful, though the writer has several times attempted to take advantage of it without any success, and aside from this believes the method objectionable.

A CASE OF SYMPATHETIC OPHTHALMIA; OPERATION ON THE EXCITING EYE, WITH RESTORATION OF VISION IN BOTH.

By Dr. JOHN F. FULTON, Ph. D., St. Paul, Minn.,

PROF. OF OPHTHALMOLOGY AND OTOLOGY IN THE MINNEAPOLIS MEDICAL SCHOOL.

THE subject of sympathetic ophthalmia has been introduced into medical literature quite frequently lately; by papers read in a number of medical societies and contributions to the numerous medical journals. These productions show that the profession are not satisfied either with the theories regarding the pathogeny of this disease or with its treatment. This being the condition of affairs, the publication of faithful and accurately kept records of individual cases still remains an important task, as only through such can a union of opinion be obtained on disputed points.

Out of this confusion of controversy comes a ray of light which seems to be growing wider with extended experience and investigation, viz.: that enucleation is not so frequently necessary as was formerly supposed, the sympathetic trouble being capable of being relieved by less severe measures. It is in support of this latter statement that the following is submitted for publication.

A Swede, G. A. P., aged twenty-six years, was referred to me for treatment from a neighboring town in Wisconsin. He reported at my office on the 20th day of Jan., '84, with this history: Six weeks previously, while at work in the woods, was hit in the left eye with a sharp stick. His vision was considerably injured at the time of the accident; had been treated by various solutions being dropped into the eye, and the application of the bandage;

had noticed that within the last few days the vision was becoming "cloudy" in the right eye, and that this eye gave him some pain during the night. An examination showed the *status præsens* to be a large cystoid cicatrix extending in the vertical meridian, at the sclero-corneal junction, near the outer ciliary body. The prolapsed iris was imprisoned in the cicatrix, giving rise to a pear-shaped pupil and narrow anterior chamber. The lens was not injured; the cornea hazy; marked ciliary injection and irritation of conjunctiva; profuse lachrymation and great dread of light; interocular tension diminished; some tenderness revealed by pressure over the ciliary region; ophthalmoscopic examination could not be made; vision $\frac{20}{200}$ (reads, J., No. 13 at 8 in.).

The condition of the R E was that of the stage of irritation, but more. In addition to the photophobia, lachrymation, slight conjunctival irritation, and weakness of the accommodation; the iris was slightly discolored and swollen, and acted slowly. The application of atropine revealed no synechiæ, but it was with great difficulty that the pupil could be dilated ad maximum; a few small opacities could be seen on the posterior part of the cornea by the aid of oblique light and the ophthalmoscopic illumination; the media were quite clear; the disc red and swollen, its outline could be made out with difficulty; the veins were full, broad, and tortuous; small whitish streaks of exudation could be distinctly seen along the course of the blood-vessels in various parts of the retina, thus proving that we had a case of neuro-retinitis to deal with.

The patient being extremely anxious to have his eye spared, saying that he would rather suffer death than the loss of an eye, and remembering the words of Mauthner, "that iridectomy on the eye causing sympathy is only to be done when the iris has been incarcerated in the the peripheral wound in the cornea, for then we may succeed in saving both eyes," I determined to try first an iridectomy.

The operation was performed without ether on the 21st of January. A large piece of the iris was removed at the seat of prolapse; there was but slight hemorrhage into the anterior chamber; sol. of atropia, 1 : 125, was dropped into the eye, and the bandage applied. The sympathizing eye was treated with atropia, rest in a darkened room, and by the administration of corrosive sublimate ($\frac{1}{10}$ gr. three times daily).

Jan. 26th, left eye in excellent condition, wound healed perfectly, and the pupil in proper position ; anterior chamber good ; cornea clearing. In a few days the R E showed well-marked signs of improvement. The opacities on Descemet's membrane disappeared ; the infiltration of the retina became absorbed, and the outline of the disc became more and more sharply defined, and soon resumed its normal level, and vision improved from $\frac{20}{40}$ (at the time of the first examination) to $\frac{20}{30}$, and finally the patient was dismissed a few weeks later with perfect sight in this eye, with only a slight venous hyperæmia, and here and there along the blood-vessels a few whitish streaks to give evidence of his former trouble. The most careful scrutiny failed to give any evidence of atrophy of the optic nerve.

The improvement after the operation was so rapid that there is but little doubt as to its being the means of bringing about the good result, although a case of a similar nature was reported in these ARCHIVES by F. Samelsohn (*A. of Ophthalmology and Otology*, vol. v.), which terminated favorably and no operation was performed at all. In Samelsohn's case the injury to the exciting eye was much more severe than it was in the case which I have reported, and in which the sympathetic trouble showed itself in the form of serous iritis.

Mr. Simeon Snell reported a case (to the Ophthalmological Society of the United Kingdom) of plastic iritis caused by a sclero-corneal wound of the fellow-eye, and prolapse of iris, which was perfectly relieved by excising the prolapsed iris and thus removing the source of irritation (O. R., Aug., 1883). The patient resumed his work in a few weeks after the operation with V $\frac{20}{30}$ in either eye.

DESCRIPTION OF AN EYEBALL AFFECTED WITH COLOBOMA OF THE IRIS AND CHOROID.

By Dr. DA GAMO PINTO.

ASSISTANT AT THE OPHTHALMOLOGICAL CLINIC AT HEIDELBERG.

(*Plates ii. and iii. of Vol. xii., German edition.*)

Translated by Dr. M. L. KING, New York.

THE eye to be described in this paper, was enucleated in May, 1882, by Prof. Hirschberg, because of threatened cyclitis, and it has already been mentioned in the work of Otto Becker.[1] If I refer to it again, it is not only because of the relative scarcity of microscopic investigations of coloboma of the iris and choroid, but principally because this eye presents other changes which seem to explain or clear up some other pathological processes.

History.—The following facts are taken from a letter of Hirschberg's, and are also found in Becker's book.

Th. Sch., is one of five children of healthy parents. One of her brothers also has coloboma of the iris, choroid, and optic nerve. With it there is oscillatory nystagmus, convergent strabismus, and amblyopia of the left eye. The anterior edge of the depression caused by the coloboma is very abrupt, undermined, and surrounded by a peculiar formation similar to the collar of a shirt. The atrophy of the choroid or the decoloration, extends forward, about the diameter of the disc, beyond the edge of the depression. After the patient had been treated at the out-patient department for some months, she was, on May 31, 1882, received into the hospital for enucleation of the eye, because of the excessive pain

[1] "On the Anatomy of the Normal and Diseased Lens," p. 136, fig. 61.

caused by it. Her defective vision was congenital, but in other respects she was healthy and strong. In the left eye there is a bridge-shaped coloboma of the iris, and also coloboma of the choroid. On the capsule is a delicate brownish deposit (membrana pupillaris perseverans). On examining it with a lens, it appears to be a thin, velvet-like layer, composed of fine dots and lines. It does not interfere with ophthalmoscopic examination. The lens is entirely transparent, excepting a small point in the posterior cortical layer. When the visual axis is directed forward, nearly the whole coloboma is outside the limit of the lens. The latter is decreased in size, and its lower margin is displaced upward and backward. The left eye can count fingers at 4–5 feet, and with + 6 reads Snellen II at $1\frac{1}{2}''$. In the upper part the visual field is defective. For the past year the right eye has been amaurotic, is not sensitive externally, but quite sensitive to pressure ;—T $\frac{1}{2}$. Cornea clear. Inferiorly there is coloboma of the iris. The tissue of the iris is normal only at the periphery, the pupillary part being atrophied, and composed mainly of the uveal layer. There are several synechiæ. The lens is semi-transparent, distinctly thickened, and detached from the zonula. If the patient looks toward the median line, the edge of the dislocated lens can be seen for a considerable distance. The vitreous humor is either infiltrated with blood, or entirely vascularized. A bladder-like figure is also visible.

After the enucleation on May 31st, the eye was put in Müller's fluid, and on Oct. 21st it was divided perpendicularly through the middle of the coloboma of the iris, embedded in celloidin, and cut with the microtome.

Macroscopic Description.—In the sagittal direction it measured 23.5 *mm.*, in the vertical 19.5 *mm.*, and in the transverse 20 *mm.* Externally no symptoms, such as thinness of the wall or change of color, which indicate the presence of a coloboma of the choroid. In the upper half the curve of the bulb is quite normal, but in the lower half there is a staphylomatous bulging, beginning at the optic nerve and then gradually decreasing anteriorly. Just behind the ora serrata there is a slight retraction, and here a second (but not so marked) bulge exists, extending to the sclero-corneal junction.[1] The anterior chamber, which is of

[1] These anatomical appearances correspond pretty well to the ophthalmoscopic appearances Hirschberg saw in the eye of patient's brother.

normal depth, is filled, particularly at the periphery, with a coagulated whitish exudation. In the pupillary part the iris is somewhat thickened, and at the periphery it is attached to the cornea. The lens is pushed downward, is thicker below than above, and has become attached to something behind it. Because of the agglutination of the iris to the cornea and the displacement of the lens, the upper portion of Petit's canal has become so much dilated that it forms a wide space (square on section), which is also filled with the whitish exudation. Behind the lens and attached to it and to the ciliary body is a yellowish resistent mass, which is also connected with the detached retina, and from its shape recalls the detached vitreous. The posterior part of the space, usually occupied by the vitreous, is divided into two chambers; the lower and smaller one contains the coloboma of the choroid, and corresponds to the staphylomatous bulging of the sclerotic already mentioned. The coloboma begins at the lower pupillary margin, appears whitish, uneven, covered by a fibrous membrane which is united to the sclera, but on section can be distinctly defined. Toward the anterior part this covering becomes thicker. Just behind the ora serrata it is, as well as the choroid, torn off from the sclera, and passes into the yellowish mass found in the anterior segment. This spot, where the detachment begins, is also the anterior edge of the coloboma, and corresponds to the retracted part seen on the outer side of the bulb. We find, therefore, that in the region of the coloboma the choroid is wanting, and the retina also, if the whitish covering of the colobomatous depression does not represent it.

In the remainder of the fundus of the eye, the retina is totally detached, and has shrunk so that only an irregular fibrous cord remains. This forms the boundary line between the two chambers described, and is connected posteriorly with the optic-nerve entrance, and anteriorly with the yellowish mass. Besides the shrunken retina, the posterior part of the vitreous chamber contains a whitish friable exudation, and masses of cholesterine plates. The choroid is adjacent to the sclera, excepting at that part of the ante-

rior chamber where the bulging of the sclera is found. The sclera is of about normal thickness all over, even in the region of the coloboma.

Microscopic Appearance—Coloboma of the Choroid.—Surface specimens could not be made, because of the density of the covering, and its intimate relation to the sclerotic. After teasing it, a small piece of it was found to consist of fibrous connective tissue with numerous round and oval cells, some vessels, and a few pigment-granules. By this method of preparation, the arrangement of these various elements could not be made out, but sections made in various directions were very instructive. The whole region of the coloboma is covered by a fibrous membrane, (cicatricial tissue or intercalary membrane of author's) connected posteriorly with the optic-nerve entrance, and laterally and anteriorly with the choroid, retina, and the vitreous humor. Although of varying thickness, this membrane adapts itself to all the irregularities of the sclerotic, filling the niche-like depressions. It is composed almost exclusively of connective tissue. In the vicinity of the optic nerve this tissue is more of a fibrillated character, and contains more cells than at the periphery. The interlacing bundles of fibres enclose larger and smaller spaces, filled with a homogeneous coagulated fluid (œdema). The cells, which are mostly branched, are rich in protoplasm, and have an oval or rod-like nucleus. This tissue contains but few vessels, which are derived partly from the sclerotic, and partly from the central vessels. Toward the periphery the intercalary membrane is of denser consistence. The bundles of fibres are larger, and become more parallel in their course, like those of the sclera, but are easily distinguished from the latter by their numerous nuclei. Embedded between the fibres, we see here and there amorphous pigment-granules and pigment-cells. The latter are round, oval, or irregular, and contain a round nucleus, which stains readily. They are not arranged in any regular order, as they are found in the superficial and also in the deeper layers, but they are certainly more numerous in the posterior segment of the intercalary membrane. Nothing definite can be said

as to the nature of these cells, but it must be noted that they are similar to proliferated pigment-epithelium. The fact that in the anterior part of the cicatricial tissue a colloid excrescence is embedded, surrounded by numbers of such cells, would indicate the epithelial nature of the pigment-cells.

In the periphery of the coloboma the thickness of this covering gradually increases. At the anterior margin it is 5–6 times thicker than in the centre of the coloboma.

If we consider the edges of the coloboma and the relation of the connective-tissue membrane to the neighboring parts, it will be seen that there is no well-defined margin. The pigment epithelium disappears very soon, after forming a small patch of proliferation. But the choroid can be traced to some distance into the region of the coloboma, and passes so gradually into the cicatricial tissue that no histological separation can be found. At the boundary line the connective-tissue fibres contain between them spindle-shaped, branched, pigmented, and colorless cells, which undoubtedly belong to the choroid. Masses of amorphous pigment are also found. The superficial layers of the intercalary membrane are connected with the folded retina. Histologically, it cannot be decided where the cicatrical tissue ceases and the retina begins, because the latter is so much degenerated that only the connective tissue and œdematous spaces can be distinguished. Anteriorly, there is also a well-defined connection between the covering of the coloboma and the degenerated vitreous humor. The bundles of the connective-tissue fibres grow in a fan-shaped manner, forming long, fine, divergent rays, which are gradually lost sight of in the fibrous, degenerated vitreous humor.

The posterior margin of the coloboma extends to the optic nerve. After the fibrous layer of the optic nerve has passed the lamina cribrosa, it bends as usual and covers the region of the coloboma, but it has entirely lost its nervous character, and now forms a large-meshed connective tissue layer, rich in cells. If the membrane be traced from any one point, we come to the lamina cribrosa without

noticing any dividing line. At the point alongside the optic-nerve entrance, where normally the choroid begins, there are some masses of pigment and pigmented cells, to indicate a rudimentary choroid; while, on the other side, above, the choroid begins as usual. At this point no trace of the retina is to be seen.

On the internal upper surface of the intercalary membrane, no covering is noticed, such as Manz[1] found, and thought it represented the continuation of the limitans interna. On the inner surface the connective-tissue layers become thick and are in immediate contact with the exudation filling the vitreous chamber.

Large numbers of fibres pass from the deeper layers of the cicatricial tissue to the internal surface of the sclerotic forming quite a firm connection. In the region of the coloboma the sclera is not thinner than usual, the bulging seen externally being mostly due to a curving of the fibres of the membrane. These fibres are more wavy than usual, and near the optic nerve they are so tangled up that it reminds one of the results left after the cicatrization of penetrating wounds. This spot is also noticed because of its richness in cells, vessels, and nerves. In the outer layers of the sclera, in the vicinity of the optic nerve, we find islands of fine, fibrillated cellular connective tissue, hardly distinguishable from the intercalary membrane.

If we now review the structure of the coloboma, we find that it contains neither retina nor choroid. As in many cases investigated lately, these coats are not present even in a rudimentary condition, but are replaced by a slightly vascular connective-tissue layer, which is continuous with the optic nerve, retina, and choroid. In it there are pigment cells, probably of epithelial nature, and a colloid formation. The sclerotic is not attenuated, but in places there is great irregularity in the disposition of its fibres, and a greater number of cells, vessels, and nerves than normal. In the outer layers of the sclera there are isolated masses of fibrillated connective-tissue.

If these histological results are sufficient to form an

[1] *Klinische Monatsbl. f. Augenheilk.*, 1876, p. 8.

opinion as to the origin of the coloboma of the the choroid, we would support the view usually accepted. From embryological and pathologico-anatomical investigations, it is assumed that this deformity is caused by a non-closure of the fœtal fissure of the secondary ophthalmic vesicle, resulting in defective development of the outer parts of the eye. The edges of the fissures are connected later by newly-formed connective tissue, the latter being developed from the cephalic plates surrounding the embryonal nuclei of the eye.

As the fœtal ophthalmic fissure has reference particularly to the retina, and because retinal elements are found in the coloboma, attempts have lately been made to upset the view, that this defect in development is due to the retarded and incomplete closure of the retinal fissure. In three cases only which have been subjected to microscopic examination —and such only can be considered—has it been shown that retinal elements have been present. In Arlt's[1] case the retina, as well as the choroid, was not defective, though it was present only in an atrophied condition. The retinal elements were, as Arlt expresses it, "drawn asunder and loose." The second observation is that of Manz, who investigated a case occurring in the ophthalmic clinic at Rostock. In the cicatricial tissue he was able to demonstrate the presence of isolated masses of retinal elements, which were however considerably changed in structure.[2] Of much geater interest is the case published by Haab.[3] He found the whole extent of the coloboma covered with retina which, in the anterior part, was even thicker than normal. The typical arrangement of the various layers was not present however. He says, *loc cit.*, p. 261: "The materials for the formation of a retina are present; both granular layers, the layer of ganglion cells, the molecular layers, etc., are here, but in great confusion; the granular layers are taking a wavy course, then again forming rings,—in the middle of which are the limitans externa and the rods and cones—or there are isolated spots formed of granules, with rods and cones in the centre of the retina," etc.

[1] Arlt: "Krankheiten des Auges," Bd. II., p. 129.
[2] *Klin. Monatsbl. f. Augenheilk.*, 1876, p. 1.
[3] *Archiv f. Ophthalmol.*, Bd. xxiv., 2, p. 257, *et seq.*

From these investigations we find, that if retina be present at all, in the region of the coloboma, it is either in disconnected parts, or its elements are thrown together in disorder. It is therefore safer to assume, as Manz[1] has already done, that even in these irregular forms, the development of the coloboma depends on a retarded closure of the embryonal ocular fissure, with the distinction that the tissue filling the fissure, by its contraction and expansion, draws parts of the neighboring retina into it. This view is supported by the folding and thickening of the retina in Haab's case.

The cause of the retardation of the closure of the cleft, is of much importance for us. In his work on deformities of the eye, Manz[2] has advanced the supposition that such a change in the development might be caused by the bending in of the cephalic plates into the cavity of the secondary ocular vesicle, this bending in, being caused by the entrance of the vessels into the interior of the eye. Deutschmann advances another view.[3] Two years ago he had the opportunity of examining a coloboma of the iris and the choroid, in the eye of a rabbit, and thought he could replace the vague notions on arrest of development by an explanation based on facts. In the region of the coloboma he found symptoms of a sclero-choroido-retinitis which had not yet disappeared, and he connected this fact with the origin of such malformations. From his point of view therefore, a coloboma is nothing else than the result of the disturbed development of the eye, brought about by some intrauterine inflammation. Hölzke[4] who has also examined such a case, has lately also supported Deutcshmann's view.

In the case under consideration there is no objection to assuming an intrauterine inflammation, which brought about the coloboma. The peculiar conditions present in the sclerotic, the tangled fibres, the abundance of cells and

[1] *Archiv f. Ophthalmolog.*, Bd. xxiv., p. 20, u. Nagel's Jahresbericht, 1878, p. 202.

[2] Graefe-Saemisch, *Handbuch d. ges. Augenheilk.*, Bd. ii. Cap. vi. p. 81.

[3] *Klinische Monatsbl. f. Augenheilk.*, 1881. p. 101.

[4] These ARCHIVES, xii., p. 175.

granules, the great vascularity, and the isolated masses of fibrillar connective tissue, might all be supposed to be the remains of an inflammatory process. Although these changes might with great probability also be assumed to be due to closure of the fœtal fissure (without inflammatory symptoms) by a new growth from the cephalic plates, we must not, at the present stage of our knowledge, give up belief in a lack of development, since in it we have an explanation, which will apply equally well to all malformations (hare-lip, cleft-palate, coloboma of the lids). The cause of this arrest in the normal development is just as much of a riddle as is the cause of the inflammation found by Deutschmann.

In this respect, a case observed by Pause [1] is of value. In a girl who died at birth, there was found a total coloboma of the iris, downward, and choroidal coloboma, which could be seen both with the naked eye and the ophthalmoscope. The microscopic structure of the sclera, choroid and retina, was found to be normal, the pigment-epithelium only, at the coloboma being deficient in pigment. In this case, according to Pause, there was a "partial leucosis of the pigment-epithelium." [2] It is, nevertheless, very probable that this case may be classed as a coloboma of the bulb, because of the presence of the fissure in the iris. It is evident that it is due to the persistence of a fœtal condition of some very early period of development; but that this arrest of development is due to an inflammation is, to say the least, very doubtful.

Sclerotic; Choroid.—External to the coloboma the sclerotic is not materially changed. The thickness, course of the fibres, distribution of the cells and vessels, are nearly normal; at the corneo-scleral junction there is a certain abundance of cells and vessels, probably due to some antecedent inflammation. More marked still is the pigmentation of the sclera around the optic-nerve entrance. The pigment is seen in large black or dark-brown masses which on closer

[1] *Archiv f. Ophthalm.*, Bd. xxiv., 2, p. 84.

[2] Dr. Becker assures me, that in the pig it is not at all rare to see such formations which represent choroidal coloboma. In these cases, however, the choroidal stroma is also wanting in pigment.

inspection seem to be collections of cells. The latter are large, stellate, long, or oval, and have a distinct nucleus. They are situated partly between the fibres of the sclerotic, but they are mostly along the vessels and in the tunica adventitia. Occasionally vessels are found surrounded with pigment, which can be traced to some distance in the choroid, showing the transition of the scleral into the choroidal pigmentation. Around the limbus of the cornea is a slight hyperæmia; otherwise it is normal.

As a rule the choroid adheres to the sclerotic; in the anterior segment only is the choroid raised from its supporting membrane; this spot corresponds to the place where the yellowish mass was formed behind the lens. This detachment is not artificial, and is evidently caused by the tension exerted by the changed vitreous humor and the cyclitic products. In structure, the choroid contains considerable pigment, there is some thickening of the lamellæ of the stroma, and great congestion of the capillaries. At various places the methods of preparation have separated the parenchyma into fine lamellæ, showing the branched cells of the stroma very well (fig. 1). In the region of the ora serrata, where the detachment of the choroid begins, and where it has even formed a *re-entrant angle* (fig. 1.), there is a small infiltrated spot, composed of round cells. This is probably the remains of a circumscribed inflammation.

Wherever the retina is present, it is detached. The pigment-epithelium is attached to the choroid, and presents distinct signs of proliferation. The young cells formed in this way, are present in small groups, situated partly on the epithelial layer, and partly in the sub-retinal exudation. As a rule they are deficient in pigment, some having none at all, and are generally of circular shape with bright nuclei. Many of these cells show peculiar pathological appearances, which will be more minutely described.

Colloid Excrescences of the Choroid.[1] From the optic nerve to the ora serrata, particularly in the posterior segment, there are seen numerous small and large colloid excrescences attached to the pigment-epithelium, being therefore in no

[1] I am greatly indebted to Dr. Benckiser for drawing figs. 3, 4, and 5.

relation to the choroid (fig. 3). They are roundish or elliptic in shape, well-defined as a rule, but some are seen having no outline; these seem to have burst, and gradually pass into the surrounding tissue, which has a similar structure. These excrescences, even the largest ones, are covered with a double and even triple layer of cells (fig. 3 *b*). The cells are large, nucleated, flattened, and, as a rule, pigmented. Some of them are round and not to be distinguished from young pigment-epithelium. In the interior of these glands, there are a great many nuclei, cell-debris, and pigment granules, the arrangement of which causes the whole to assume an almost lobular appearance. As regards the more minute structure of these growths, it is to be noted that the contents are not homogeneous nor arranged in concentric layers as has been stated, and which arrangement I have seen myself; the contents are finely granular, crystalline, and refract light strongly. In many of these growths, we may see (under high powers) fine needle-shaped shining granules (crystals?) (fig. 3 *d*) divided into groups, each group having a common centre. Where these needle-shaped formations are seen, there is very little of the granular matter left, so that it may be inferred that the needle-like masses are the result of crystallization (or a similar process) of the granular mass.

I have already had occasion to express my opinion as to the origin of these colloid excrescences of the choroid.[1] After an exact description of the histological characters of these formations, and mention of the views of their origin, I felt competent to assume a process of degeneration in the pigment-epithelium which had increased by proliferation. I had shown that great proliferation was present, and thought I had demonstrated the presence of degenerative processes in various stages of development. After I had sent in the manuscript, Prof. Leber was kind enough to call my attention to his already repeatedly expressed views (verified by A. Meyer) as to their origin.

I requested him to supplement my unintentional oversight by means of a note. I will quote his statement word for word:

[1] *Archiv f. Opthalm.*, Bd. xxviii., 1, p. 178.

"I would like to recall my explanation of the origin of the colloid thickening of the hyaloid membrane of the eye, which was based on thorough analysis of all points bearing on the subject. It has received but little attention, probably because I communicated it only incidentally (*Graefe's Archiv*, Bd. xix., 1, p. 187, 1873; Bd. xxv., 1, p. 308, 1879), though my views are supported by Ad. Meyer, who investigated this subject (*Graefe's Archiv*, Bd. xxiii., 4, p. 168). I am of the opinion that these thickenings, as well as the hyaloid membrane itself, are developed from the neighboring epithelial layer, for this supposition is the only one which will explain all the appearances satisfactorily. That these hyaline masses sometimes include pigment- and cell-debris, does not invalidate this view, because these things are also seen in capsular ataract, for instance; they are explained by the alternate deposition of cuticular layers and newly formed cells which later undergo atrophy."

After reading the above papers of Leber and Meyer, I still could not see how the assumption of a cuticular formation could be applied to my case, because I can see no analogy between the new colloid formations on the choroid, and the amyloid bodies of the conjunctiva, on which Leber's view chiefly depends. Even if the enclosure of pigment- and cell-debris in the hyaline mass can be explained by an alternate deposition of cuticular layers and newly formed cell-layers, which become atrophied later on—this still gives no explanation of the origin of the colloid excrescences. I had laid as much stress on these beginning stages as on the more advanced ones. I have not replied to these views, first, because I only saw them when reading the proof-sheets; and secondly, because at that time I had not the proof which could only be found by an investigation of the pathological changes in each cell. I think I can present such proof at present. In opposition to Leber's "unproved hypothesis," as he himself calls it,[1] I can now bring facts which have justified me in assuming that in both of my cases the formation of both of these growths depended on a degeneration of the proliferated pigment-epithelium.

As already mentioned, the layer of pigment-epithelium

[1] *Archiv f. Opthalm.*, Bd. xix., 1, p. 187.

of the choroid shows marked proliferation at various points. The newly formed cells are partly pigmented, partly devoid of pigment, but all have the same shape, size, and nucleus, there being thus no doubt as to their common nature and origin. Among these young cells there are a few which, without change of form or size, have homogeneous, light, slightly colored contents, in which the nucleus and pigment-granules are embedded as in other apparently normal cells. Beside them are other cells, in which the homogeneous character is more marked, thus leading gradually to others in which the whole protoplasm has become changed. At this stage, the cells are somewhat enlarged, swollen by imbibition, and of round shape. These cells form round homogeneous and also finely granular drops which refract light strongly. The nuclei are deeply colored, and contain a varying quantity of pigment, this depending on the quantity of pigment the cell originally contained (fig. 4, *a* and *b*). But it must be observed that the nucleus retains its original position, being completely surrounded by changed protoplasm. I have never seen the protoplasm changed in circumscribed areas; it always includes the whole contents of the cell, the different stages varying only as to intensity. There can, therefore, be no dropwise secretion of cuticular substance within the protoplasm, a kind of endogenous new formation by the formative action of the cell, such as Leber assumes for the amyloid bodies. Even under the highest powers, I have, in these cases, not been able to discover a membranous nucleated capsule, such as is seen in the amyloid bodies.

Later on the nucleus also becomes degenerated. Besides the sharply defined, deeply colored nuclei, there are others becoming paler and paler (fig. 4 *c*), until at last they are distinguishable only as cloudy, bluish, or reddish discs (fig. 4 *d*). Other cells contain only the remains of a nucleus or perhaps none at all. We have thus gradually come to the smallest, most rudimentary growth which presents itself as a round, homogeneous or finally granular drop, with or without pigment-granules (fig. 4 *e*. *f*). By coalescence of these drops the larger growths are formed, which, as all know, may

assume great proportions. This process may be observed in almost any preparation. In the midst of the group of the degenerated but isolated cells, drops are seen, which are larger, round or irregular in shape, with two, three or more nuclei and a varying quantity of pigment (fig. 4, *h. z*). The nuclei are partly normal, while others are partly or totally degenerated. In such groups we occasionally succeed in making out the outline of the individual cells, which are flattened against each other (fig. 4 *g*). Of peculiar interest are the larger groups of cells which are seen on the pigment-epithelium, and have the shape of a fully developed excrescence (fig. 5). In these groups we may see at once all the various stages of development I have described: single well-defined cells in various stages of degeneration; double or triple cells, larger groups, the protoplasm of which has coalesced, the cell-borders being indicated by light lines; and lastly, large granular masses, containing cell-debris and pigment-granules, similar to a fully developed excrescence.

From the result of these investigations I should not only feel justified in still supporting my view that these colloid excrescences of the choroid originate in a degeneration of the proliferated pigment-epithelium, but I will be more exact, and state that this degenerative process takes place in the protoplasm of the cell, involving the nucleus secondarily. We therefore see that the formation of these excrescences can in no manner be compared to a cuticular secretion, because the latter represents a *formative* and the former a *regressive* process.

I cannot now say whether the products of this degeneration are hyaline or not. In a recent work [1] v. Recklinghausen assumes such to be the case.

The Optic Nerve.—In the optic nerve very extensive changes are to be seen, which, as a whole, may be called a process of sclerosis. The external sheath presents nothing characteristic, but the inner sheaths show well-marked changes. The trabeculæ of the arachnoidal sheath are

[1] "Handbuch der algemeinen Pathologie des Kreislaufes und der Ernährung," p. 407.

enormously thickened, infiltrated with cells, and refract light strongly; they stain very badly, and have a peculiar homogeneous appearance. Similar appearances are also to be seen in the pial sheath, but in this the infiltration of cells is most prominent. These two sheaths are about five or six times the normal thickness. In the trunk of the nerve, also, the connective-tissue trabeculæ are to be seen thickened and brighter than usual. Particularly prominent is the central cord, which, in longitudinal sections, appears as a broad, light, finely-fibrillated band, deficient in cells. The walls of the central vessels are unchanged. At the lamina cribrosa, the increase of connective tissue is still more marked; here the enlarged trabeculæ form a dense shining network, the meshes of which are hardly to be distinguished. The position of the lamina is normal. The optic disc has become changed into connective-tissue, but is still connected with the shrunken retina. After the optic-nerve fibres have passed through the choroid, they assume an antero-posterior direction, parallel to one another, are of a fibrous nature, contain fewer cells, and hide the central vessels, which seem to be normal.

A peculiar formation, found in the lower segment of the papilla of the optic nerve, is of considerable interest. Just at the point of meeting of the scleral and choroidal portions of the optic-nerve entrance, there is a slight excavation in the substance of the sclerotic, which is filled by a finely fibrillar, very cellular connective tissue. This tissue is connected with the remainder of the papilla optica, but is at the same time so markedly of the same structure as the membrane in the region of the coloboma, that it is not unlikely that they have a common origin, especially as they are only separated by a narrow bridge of scleral tissue. If this excavated part of the optic nerve be examined by an unprejudiced eye, it might be considered to be a shallow glaucomatous excavation which had become filled with connective tissue; but this supposition is at once shown to be fallacious, because the excavation is only present in the lower part of the nerve, corresponding to the coloboma. It is therefore probable that it is a rudimentary coloboma of the

optic nerve, which had become closed by a process similar to that of the coloboma of the choroid. The finely fibrillar connective tissue within the scleral excavation would then be called cicatricial or intercalary tissue.

Retina.—Attached to the degenerated nerve-trunk we find the detached retina which is thrown into folds and, with the vitreous humor, forms the mass seen behind the lens. Near the ora serrata where the true retina ends, and its attachment to the choroid becomes closer, the tension has become so great that the choroid is also detached from the sclera. We have already mentioned the angle formed at this point and its division into lamellæ. Next to nothing of the structure of the retina is to be found. The retina seems to be converted into connective tissue, and besides the œdematous spaces filled with coagulated exudation, contains large numbers of nuclei. Part of the latter are oval or rod-like, and there is no doubt as to their connective-tissue nature, while others are round and by their arrangement call to mind the granular layers of the retina. Between the normal, easily stained nuclei, there are others presenting peculiar changes. On the whole they have preserved their original shape, but are swollen in a peculiar manner, seem homogeneous, and stain less readily than those which seem normal. A similar homogeneous consistency is also to be observed in the thickened walls of some of the finer vessels; the nuclei being well-preserved, and quite marked by their deep staining. In this connective tissue are seen pigmentations scattered or gathered in groups; they are formed partly by amorphous granules, and partly by cells containing pigment (pigment epithelium). Of great pathologico-anatomical importance are the changes found in the ciliary part of the retina. We find here symptoms of proliferation not only in the pigment-epithelium but also in the epithelial-like cylindrical cells. The newly formed cells are of epithelioid character, are large, rich in protoplasm, may or may not be pigmented, and have a round or oval nucleus. They either compose large groups, covered with scattered pigment, or long cones, or they may be in the form of a net-work extending into

the vitreous chamber, enclosing in their meshes, changed vitreous humor or coagulated exudation. This net-work is in connection with the folded retina and assists materially in the formation of the mass lying behind the lens. In some parts the cylindrical cells of the pars ciliaris retinæ have developed into long narrow rods (with long nuclei) which form a single or several layers, just as proliferated cells do, retaining at the same time their round shape; cone-like projections may also be formed. Many of these long cells are arranged in double rows, so disposed that their ends touch, and with the other double rows forming an alveolar structure, which reminds one of the arrangement of a cylinder-celled carcinoma.

In the present case, no fibrous axial tissue is seen, around which these double rows might have been formed; the cells are all in close proximity to each other. It is therefore not improbable that this columnar arrangement of cells results from a local cellular hyperplasia, and is favored by a tension from within, of which we will speak directly. Be this as it may, it is certain that these cells are of an epithelioid character, and undoubtedly originate in a proliferation of the pars ciliaris retinæ.

The Ciliary Processes.—Proliferations similar to this and to the same degree are also seen in the ciliary processes, which seem to become elongated and spiral in shape (fig. 1). The proliferation includes the epithelial layer, which, as is well known, is considered as remains of the retina, as well as the uveal layer of the ciliary processes. The proliferated cells, all presenting the same type, in part contain pigment and cover the points of the ciliary process in large irregular masses. The regularly pigmented rows, which normally show so well in sections of the ciliary processes, are scattered in these cases, and gradually become lost among the masses of non-pigmented proliferated cells. The proliferation is most marked in the lower segment of the bulb, where the dislocated lens flattens and presses the ciliary processes backward.

As regards the pathologico-anatomical importance of this extensive cell-proliferation, which includes the whole ciliary

region, I am inclined, from my observations, to connect it with a cyclitis, thus assuming it to be the result of an inflammation of the ciliary body. I have had repeated opportunities of seeing such appearances in eyes with cyclitis. The so-called cyclitic pseudo-membrane consists mostly of long rows or networks of cells, all arising from a proliferation of the epithelial elements of the ciliary region. My colleague at the Heidelberger eye-clinic, Dr. Schæfer, has also expressed this view in his late paper on "Traumatic Iridodialysis and Irideremia."[1]

The Vitreous Humor.—Within the narrow and very irregular cone, formed posteriorly by the detached retina, and laterally by the pars ciliaris, is found the remainder of the changed vitreous humor. It is mostly finely fibrillated, containing comparatively few cellular elements and pigment, together with some medium-sized and fine vessels arranged in networks. In many preparations it can easily be shown that these vessels originate in the ciliary body. The remains of the vitreous humor are closely connected with the surrounding tissues by bundles of fine nucleated fibres, refracting light strongly; these pass from the degenerated retina into the fibrous vitreous humor, and are gradually lost. These relations are particularly well shown at the anterior edge of the coloboma, where the intercalary membrane, increasing in thickness, rises above the pars ciliaris like a wall. This anterior margin of the coloboma now divides into numberless small bundles, which radiate anteriorly and laterally, thus forming an intimate connection between it and the vitreous body. Occasionally such nucleated extensions can be seen between the cells of the pars ciliaris. Just behind the lens, the vitreous humor (or the newly-formed body?) assumes a hard, fibrous appearance. The fibres are parallel to the surface of the lens, being so closely connected with the folded retina behind and the posterior capsule in front, that it is very difficult to choose a histological boundary line between these different tissues (fig. 1). Below, but still connected with the capsule, this fibrous tissue curves around the equator of the lens,

[1] *Archiv f. Ophthalm.*, Bd. xxix., 1, p. 21.

and forces itself between the displaced lens and the flattened ciliary processes (this flattening being caused by the coloboma of the iris) into the anterior chamber. Here it divides into two parts: one, the largest, covers the end of the coloboma of the iris, and forms attachments to the membrane of Descemet and the space of Fontana; the other passes up along the anterior capsule, where, after spreading in all directions, it ends. In sections of the lens, this second part of the fibrous mass seems to be a fibrillated deposit on the outside of the anterior capsule. It is but sparely supplied with vessels and cells.

The whole contents of this cone, together with the proliferations of the pars ciliaris and the ciliary processes, is probably nothing else than the so-called cyclytic pseudomembrane, the structure and origin of which are very rarely touched on in ophthalmological literature. During life this cicatrix must have possessed such an immense tensile power that it was possible for it to detach the choroid from the sclera and to luxate the lens. I will remind the reader of the reëntrant angle formed by the choroid near the ora serrata, and also of the displacement of the lens.

Ciliary Muscle and Iris.—There is nothing peculiar about the ciliary muscle except a considerable pigmentation and slight cellular infiltration.

The coloboma of the iris is total, and extends downward, so that on section we get a good view of the point of attachment of the iris to the anterior side of the ciliary body. This point is connected to the lower edge of the lens by means of the proliferating pseudo-membrane, which extends into the anterior chamber and is so highly pigmented that only large black masses are seen, the detailed structure of which cannot be made out even in thin sections. There seems to be no atrophy of the iris present, but the iris has become developed to an extent rarely seen in normal eyes. The extraordinary thickness and breadth of the sphincter of the iris is particularly marked; it may be considered to be hypertrophied. Laterally and below, in fact wherever a portion of the iris can be seen, there is a rudimentary sphincter also. This would correspond to

what is usually found in coloboma of the iris, that the small ring of iris can be followed along the margins of the coloboma to the periphery.

In the periphery of the anterior chamber the iris has grown fast to the cornea (about 1 *mm.*), just as happens in glaucoma (fig. 1). Descemet's membrane can be traced through this part to the sinus of the anterior chamber (fig. 2, *a*), but its endothelial layer passes on to the artificial angle of the iris (formed by the agglutination of the iris and cornea), and extends uninterruptedly to the anterior surface of the iris, spreading irregularly (fig. 2, *b*). The pupillary edge of the iris is bent forward on itself, and has become fastened in this position by finely fibrillated connective tissue. Because of this circumstance the free edge of the iris is not pointed and wedge-shaped, as is the case normally, but it is thick and rounded (fig. 1). It is to be noted that the greatly developed posterior uveal layer of the iris does not stop at the folded pupillary margin of the iris, but is continued on its anterior surface, covering about two thirds of its extent (fig. 1). This anterior layer of pigment is as thick as the posterior one, being even thicker in places, especially where it ends. As a rule, the covering of pigment ceases where the endothelial layer (already spoken of) begins. This latter layer covers, however, the pigment in certain places. In the upper segment of the iris, at the point of junction of these two abnormal layers (pigment and endothelium), there is seen closely attached to the iris a thick deposit composed of fibrillated tissue. It contains some cellular elements and pigment, and may be regarded as the result of inflammation, particularly as it seems to be continuous with the amorphous exudation still to be mentioned.

If this peculiar condition of things be compared with Hirschberg's statement that the stroma of the iris is mostly atrophied, the uveal layer alone remaining, it will at once be seen that this abnormal pigment-layer on the anterior surface of the iris may have appeared as such an atrophy, when in reality there was a great hypertrophy of the stroma of the iris.

We must mention a slight diffuse cellular infiltration of the iris, at the periphery the cells were accumulated in groups.

Anterior and Posterior Chambers and Vitreous Cavity.—Above, the posterior chamber is deep and large, this being caused by agglutination of the cornea and iris, and the dislocation downward of the lens; the fibres of the zonula are stretched and partly torn. The iris does not touch the lens at all (fig. 1).

The anterior and posterior chambers, Petit's canal, and the vitreous space are all filled by an exudation which, when coagulated, seems to be homogeneous, and does not stain deeply. It contains but very few cellular elements (fig. 2, c). A similar exudation is found in the posterior part of the vitreous space, between the choroid and retina, and in the region of the coloboma, but this is more granular and friable. This property is due to its large percentage of cholesterine, and normal and changed pigment-cells, and partly also to a large number of red blood corpuscles. At various points one finds collections of roundish drops, which refract light strongly; these are probably degenerated exudations.

The Lens.—This description is quoted from Becker's work (fig. 1).

"The lens is more spherical than normal. On the outer side of the anterior capsule there is a pigmentary deposit, enclosed in a mass of cellular tissue. As some capillary vessels are to be seen in it, it may be considered to be the remains of the pupillary membrane, especially as some remains of this membrane are also found in the left eye, without any symptoms of an old iritis. The whole of the inner surface of the anterior capsule is covered by a fibrillated capsular cataract, which probably originated below (where the cyclitic membrane touched the equator of the lens) and then extended upward. Below, it is 0.25 *mm.* thick, becoming thinner as it passes up. At the anterior pole it is 0.08 *mm.* in thickness, while the capsule itself has a thickness of only 0.008 *mm.* In structure it is fibrillated with regularly distributed elliptic nuclei. The surface toward the lens is

not covered by epithelium, but in its thickest parts it contains quite extensive deposits of a substance (lime or colloid) which refracts light strongly, and stains but slightly. At the equator all trace of a vortex has disappeared. On both sides of the sections the capsular cataract gradually merges into the epithelial layer, which lines the whole posterior capsule. To this layer are attached numerous layers of vesicles. In the lens substance itself, the beginning cataract is present as it is seen in the eyes of children. In the periphery the fibres contain numerous vacuoles, and further toward the centre the layers separate as in diabetic cataract. (See figs. 24–26.) In these spaces we find a homogeneous substance, rolled up in round masses. At places, the lens fibres are ruptured, and this mass has divided into small, refracting granules; these are usually assumed to resist the passage of light, and thus cause the cloudiness of the lens.

"This case also indicates that the formative activity of the intracapsular cells is influenced by a vascularized deposit on the outer surface of the capsule."

HEIDELBERG, *June*, 1883.

A FOREIGN BODY IN THE VITREOUS, REMOVED BY MEANS OF A MAGNET.

By Dr. E. L. HOLMES, Of Chicago.

MR. M. D. A., twenty-five years of age, while holding a steel drill, on the end of which a comrade was striking with a heavy hammer, received a slight wound of the left eye, February 23, 1884. The local surgeon, whom he soon consulted, could discover no trace of an injury of the cornea, nor of blood about the eye or in the anterior chamber. The following day, however, a minute gray opacity of the cornea was visible.

Two days after the accident, the patient came under my care. I found the pupil moderately dilated with atropine, the conjunctiva somewhat injected, the anterior chamber of normal depth, the tension not increased. A minute opacity was apparent between the centre and lower margin of the cornea. The radii of the lens were already quite opaque, so much so, in fact, that an ophthalmoscopic examination was scarcely as satisfactory as that with the oblique illumination. By either method a foreign body, with a grey metallic lustre, could be seen trembling with the motions of the globe, in the nasal portion of the vitreous, at a small distance behind the lens. This body, evidently very minute, appeared to be five or six millimetres long, and three or four wide. Vision in all directions was very obscure ; but the field of vision seemed intact. The opacity of the lens rendered it difficult to locate the wound in the capsule. From the fact that the patient was in a dark place when the accident occurred, and that the pupil was probably dilated, there is reason to suppose the iris escaped injury.

Although there was no pain, the patient willingly consented to an attempt to remove the steel by means of a magnet. An in-

cision, a third of an inch in length, parallel with the lower border of the external rectus, just behind the ciliary region, was made through the conjunctiva. After hemorrhage had entirely ceased, an incision an eighth of an inch long was made in the direction of the conjunctival opening, through the walls of the globe. The probe of a Gruening's magnet was gently introduced in the direction of the foreign body, and carefully withdrawn. The third effort was attended with success. I was unconscious of any sensation, when the piece of metal came in contact with the magnet. The operation was completed by the excision of a small button of prolapsed vitreous, by the insertion of a couple of stitches in the conjunctiva, and by a dressing of atropin and boracic acid. Scarcely any pain or reaction followed the operation.

The piece of steel was two millimetres long, one wide, and half a millimetre thick. Contrary to my expectation, the fragment was jet black without lustre. I had recently examined an eye, deep in the fundus of which was a piece of steel which was "dead" black as seen with the ophthalmoscope. I supposed the fragment which I had removed would, at least on one side, present the appearance of a recent fracture.

I could not regard the preliminary extraction of the lens, almost normal in its condition, by Graefe's method, as a safe procedure. For the following reasons I pursued the plan above described.

I have observed three cases of traumatic cataract without irritation, in the substance of which minute pieces of steel had been carried during several years. The lens often receives minute punctures without intra-ocular inflammation as a result.

I have observed five cases of quite severe punctures of the ocular walls posterior to the ciliary region, with prolapse of the vitreous, in which the wounds healed readily after the insertion of stitches in the conjunctiva.

Years ago I performed Hancock's operation many times for various abnormal conditions without unpleasant consequences.

I made the incision in the sclerotic opposite the foreign body, because I believed the edges of the wound might suffer less violence than in any other position. I could fol-

low the magnet behind the pupil in the direction of the metal more clearly than I could have done if it had been introduced at the inner angle of the eye.

Ten weeks after the operation the eye was entirely free from irritation and sensitiveness. The pupil was normal ; the cataract not mature—so far from it that the hands of a watch can still be seen. The patient was able to perform his ordinary duties without discomfort.

THE INDICATIONS AND CONTRA-INDICATIONS OF JEQUIRITY.

Second communication to Dr. H. KNAPP from Dr. L. DE WECKER.

I AM greatly obliged to you for the review of this question in the last number of your ARCHIVES, as this is undoubtedly the surest way to obtain an insight into the action of the remedy, and proves that you are earnestly endeavoring to arrive at the truth.

The review published in the last number of your ARCHIVES (vol. xiii., No. 1) would have been entirely favorable to the remedy had you not published two cases in your supplement, which, on account of the perforations of the cornea, not only lowered your expectations of the future of jequirity, but may do the same with our colleagues who are not yet familiar with its use. You will therefore readily understand why I shall try to discover whether jequirity is really to blame.

For this purpose I must recur to the physiological action of jequirity, which has been so extensively tested both on man and animals that a discussion would seem superfluous. The jequirity ophthalmia takes a perfectly regular course when the remedy is applied to a normal conjunctiva. If a severe inflammation has been produced, so that the conjunctiva is covered with a diphtheritic membrane, while the lids are hard and cannot be everted, the detachment of the membranes at the edges of the lids does not begin until the third or even fourth day, and if they are removed with forceps, fibrinous membranes of such density may be repro-

duced upon the upper lid as to resemble an artificially exposed tarsus. On the fifth or sixth day the œdema of the lids rapidly disappears, the membranes are no longer reproduced, while a slight swelling of the conjunctiva, which may be covered with a thin mucous coat, can persist for several days, but in the majority of cases the conjunctiva appears normal again two to three weeks after a single intense application of jequirity, except perhaps a slight swelling in the fornix in cases which have been detained too long in the house.

There never is any purulent secretion the whole time the jequirity ophthalmia lasts; a thin, watery discharge trickles down over the cheeks, while a whitish viscid substance covers the edge of the lids. Jequirity, therefore, does not produce a purulent, but a croupous or croupo-diphtheroid, ophthalmia—croupous, if the remedy is only applied very superficially; croupo-diphtheroid, if an intense action has been produced by rubbing or the application of strong infusions.

Only he who, like myself, has applied the remedy in hundreds of cases to the normal conjunctiva can positively state that jequirity has a perfectly regular and constant action, that it is an ophthalmia *sui generis*, is never of a blennorhoic character, nor is ever followed by blennorhœa. If such is the case, the purulent discharge existed before the application of the remedy, and was merely increased by it.

When Prof. Sæmisch requested me in the interest of his investigations to ascertain the number of trachoma patients in my practice, I carefully compiled the statistics and found that they only constitute two per cent. Among 6000 patients treated annually there are not more than 120 with trachoma. This number would have been too small to study the action of jequirity, therefore I also applied it to a large number of patients with pustular keratitis, scrofulous pannus, and corneal sclerosis. In the majority of these cases the conjunctiva was normal, and by carefully studying them I satisfied myself that the danger of acute diphtheria or acute purulent ophthalmia can be entirely excluded.

After having thus treated more than 1000 patients with a healthy conjunctiva in the presence and under the constant supervision of the assistants and students, I may fairly assume that no danger attends the careful single or even repeated application of a three-per-cent. infusion of jequirity to a normal, non-secreting conjunctiva. You may be sure that my enthusiasm would not have sufficed to continue my experiments in such an extensive manner upon patients with a healthy conjunctiva if I had had any results similar to yours.

But it is quite a different matter, as I stated in my last letter, to treat with the infusion cases of mixed trachoma or even chronic purulent ophthalmia with marked papillary swelling. Here the irritation due to the remedy may have disastrous results, although the genuine jequirity ophthalmia has not been produced. I have particularly insisted that in such cases we must be guided by the amount of the secretion, and must never apply the remedy to eyes affected with a purulent discharge.

Of one of the patients you say: "The first in which the cornea perforated had ordinary trachomatous pannus, no discharge, no corneal ulcers"; of the other you only say, "the corneæ were but slightly opaque"; as no secretion is mentioned in the second case, I presume there was none.

You accuse the jequirity of having produced the perforations in these two cases, to which I would simply reply that according to your own statement you did not produce the genuine jequirity ophthalmia at all, as you say, "membranes marked, and when three days later they were cast off a most violent pyorrhœa followed, which in two days affected the other, not inoculated eye." I make this assertion with the greatest confidence and for the following reasons: 1. The membranes are never entirely cast off on the third day; in an intense jequirity ophthalmia they become slightly detached from the margin of the lids and can only be removed with difficulty and great pain to the patient, while a fibrinous reproduction at once takes place. 2. Under normal conditions the jequirity ophthalmia never becomes purulent or is followed by a purulent ophthalmia.

3. The fact that on the fifth day the patients inoculated the other eye with the discharge of what was supposed to be a jequirity ophthalmia clearly proves that this was not a jequirity ophthalmia at all.

Every one who has attempted inoculation knows the difficulties it meets with, and in *v. Grafe's Archives* (vol. xxx., 1, p. 270) I have set forth the reasons why spontaneous inoculation is never observed and why it differs so much from purulent ophthalmia. Your patient inoculated his other eye with the secretion of the purulent ophthalmia which had been intensified by the application of the jequirity.

Your two cases, therefore, again prove how careful one must be in the use of jequirity in purulent conditions of the conjunctiva or in patients so disposed, but are not an argument against the application of the remedy in dry granulations or cases of normal conjunctiva in which there is no discharge of pus.

Allow me also to touch upon another point in your interesting paper. You conclude, thirdly, in your supplement that "the cure of trachoma by jequirity, as well as by nature and other remedies, is accompanied by more or less atrophy of the conjunctiva and the formation of cicatricial tissue." From the beginning of your paper it is evident that you fear that the croupous and especially the croupo-diphtheroid jequirity ophthalmia is always accompanied by the formation of scar-tissue; the question of such danger can, however, hardly be solved by one who has treated only trachoma with jequirity, as it is here almost impossible to determine how much of the cicatricial tissue is due to the original disease and how much to the remedy. The case is different, however, when one has had the opportunity like myself to examine hundreds of patients whose conjunctiva while normal had been treated with jequirity. If the inflammation is not severe, not the slightest trace of it in the shape of scars remains; if it is intense and of long duration, the scars are only faint whitish streaks upon the conjunctiva of the tarsus, as is also stated by Sattler; they have been observed in cases of sclerosis due to parenchyma-

tous keratitis, in which the inflammation had been pushed to a maximum intensity, so that the eye could not be inspected for several days.

Those who have treated many cases of trachoma with jequirity will agree with me in claiming as one of the greatest advantages of the remedy, its power to soften the cicatricial tissue already formed, a point to which also v. Hippel has called attention. The patients greatly appreciate this stretching and softening of the cicatricial tissue, as immediately after the disappearance of the inflammation, they state how much smoother the lids feel, and that the sensation of tenseness and friction has disappeared, even in cases in which the granulations persist. This is the reason why in spite of the painfulness of the inflammation the patients are always willing to submit to another application if the first is not successful, or a fresh eruption of granulations takes place.

I therefore come to a diametrically opposite conclusion, namely, that the jequirity ophthalmia not only produces the least amount of scar-tissue, but also favorably affects that which had previously existed.

As I stated in my previous letter, I attach the greatest importance to your opinion, and would regret it very much if one occupying so prominent a position in ophthalmology should obstruct the light of truth and cast a shadow upon the solution of a question of such great importance to suffering humanity.

As regards myself, I should be very exacting if I were not satisfied with the results thus far obtained in the solution of this problem. Two years have not yet elapsed since the introduction of the remedy, and already it has succeeded in banishing the barbarous method of inoculation with blennorrhoic pus from ophthalmology, and proven that it can cure the most obstinate cases of trachoma.

Would it not be absurd to claim infallibility for jequirity? Is the fame of v. Graefe's great discovery at all diminished by the fact that in a few cases iridectomy proves disastrous, and in others fails to effect a permanent cure?

You would therefore do me a great service if you would

not allow yourself to be discouraged by those two unfortunate cases, but would continue the use of the remedy; I am even now firmly convinced that if you would give jequirity another and extensive trial, you would come to more favorable conclusions than those contained in your supplement.

PARIS, *May 12th*, '84.

FURTHER OBSERVATIONS ON THE USE OF JEQUIRITY.

By H. KNAPP.

AFTER the conclusion of my paper on the use of jequirity in trachoma (these ARCHIVES, vol. xiii., p. 145) I have treated, at the New York Ophthalmic and Aural Institute, seven other cases, six with good results, one with the loss of both eyes. This last case and another one which terminated favorably were in so critical a condition when I corrected the proof of my paper, that I thought it my duty to supplement the paper by a note of warning, mentioning these cases which had shown a more unfavorable action of the remedy than had come under my observation before. In the present communication I shall not describe the five cases in which the reaction was as usual and the result good, but beg to describe in detail the two others.

CASE I.—Chas. Reynolds, æt. thirteen, sent from the Catholic Protectory in Westchester County, N. Y., had had inflamed eyes for years. When he was admitted to the hospital, Feb. 4, 1884, he showed considerable puffiness of the upper lids, and papillary swelling of the inner surface of both lids of each eye, with granular and diffuse hyaline infiltration (Stellwag's mixed trachoma). He had a good deal of photophobia; no discharge. He was treated with the copper crystal.

Feb. 11th.—Marked improvement, begins to open his eyes; corneæ clear, swelling less. This improvement continued until March 4th, when his eyes became worse; more photophobia, more swelling; lids, however, smoother. He improved again under sulphate-of-copper treatment, with cold applications several hours daily, and cleansing of the eyes with a weak solution of chloride

of sodium, or a three- or four-per-cent. solution of boric acid. The improvement by this treatment during two months had been manifest, but so slow that on *April 3d* I applied jequirity to the right eye.[1]

April 4th.—Considerable swelling and discharge; thin membranes on lids.

April 6th.—Swelling less ; membranes gone ; lids succulent.

April 7th.—Jequirity reapplied to right eye.

April 8th.—Effect slight.

April 13th.—Jequirity applied again to right eye.

April 14th.—Considerable swelling of lids ; only slight membranes.

April 15th.—Swelling increased ; muco-serous discharge ; cornea clear ; lids tense, covered with a white, even deposit which cannot be removed ; no chemosis. Cold applications ; careful cleansing.

April 16th and *17th.*—Swelling, hardness of lids, and diphtheritic deposit marked. Left eye also swollen ; free muco-serous discharge ; both corneæ slightly steamy. From the 16th iced compresses, day and night, uninterruptedly, by a day-nurse and a night-nurse. Conjunctival sac washed out every half hour with a solution of boric acid in tepid water, by means of a medicine-dropper or, when the secretion was adherent, with a camel's-hair brush.

April 18th.—Both corneæ clear. Muco-purulent discharge from right eye ; adherent deposit on inner surface of both lids of both eyes. Chemosis.

April 19th.—In right eye membranes becoming detached ; purulent discharge. Under lid ectropionized. Left eye, membranes on both lids.

April 20th.—R eye, membranes disappeared ; copious pyorrhœa. L eye, membrane being detached from upper lid, still firmly adherent to lower ; intense chemosis in outer part of conjunctiva.

[1] The application in this and other cases, after I had received De Wecker's communication, published in the preceding number of these ARCHIVES, p. 107, was as follows : three grms. of husked and crushed beans were left to macerate in 100 grms. of distilled water for three or four hours ; then the liquid was poured into a bottle, and employed in the afternoon of the same day. Two or three grms. of it were poured in a clean, small eye-cup, and out of this brushed over the everted lids with a camel's-hair brush from one to four times in succession. Nothing further was done to the eyes than keeping them clean. Repetitions of the application were made only when in two days or later the effect proved insufficient, or had exhausted itself before having produced such changes in the conjunctiva as to secure the disappearance of the granulations.

The discharge continued for two weeks; the membrane on the left lower lid disappeared April 25th; the corneæ were difficult to inspect; their lower thirds, which were easily exposed, showing no abnormity, forcible opening of the lids was not resorted to. The iced applications were continued until the patient could open his eyes, the swelling had become moderate, and the discharge had disappeared.

Patient was discharged June 12th, his eyes having felt comfortable for the last three weeks. The centre of the right cornea was slightly hazy; in the left there was a small speck near the centre of the cornea. S = $\frac{20}{20}$ in each.

CASE 2.—Wm. Godfrey, æt. eighteen, was sent from the Protectory and admitted March 28, 1884. He had chronic trachoma and pannus. The upper fornices of both eyes were infiltrated with trachoma granules, and the upper half of each cornea was vascular and opaque. V = $\frac{5}{200}$ in each eye. *March 31st*, a fresh three-per-cent infusion was brushed over the inner surface of the everted left upper eyelid. The next day the lids were swollen; there was lachrymation and moderate pain. A croupous membrane covered the upper tarsal and fornix portions. The symptoms increased for several days; the membrane was well developed and the cornea duller than before; pain slight. April 5th, the swelling somewhat less. April 6th, membrane being cast-off; conjunctiva succulent; cornea no clearer.

April 8th.—Jequirity reapplied. April 9th, effect less marked than after the first application. Lids swollen, but the ocular conjunctiva free. April 12th, lids still succulent; no membrane.

April 13th.—**Jequirity applied to upper lids of both eyes.** April 14th, considerable swelling of **right eye**, which until the 12th had been mildly touched with the copper-sulphate crystal, and was in no state of irritation when jequirity was applied. The left eye less swollen. Thin membranes on both upper lids; muco-serous discharge; slight infiltration of left cornea. Cold applications ordered. April 15th, the whole right cornea opaque, in the left a central ulcer; profuse sero-mucous discharge in both eyes. Upper and lower lids of both eyes greatly swollen, stiff, and covered with an even, white deposit, which could not be removed **(diphtheritic infiltration).** Great pain; fever. Iced compresses applied constantly, and irrigation with boric-acid solution every fifteen to thirty minutes, day and night. April 16th, the right cornea has perforated (anterior chamber empty) and a white plug,

the size of a pinhead, covers the defect. Around the centre of the left cornea, there is a ring-shaped infiltration. April 17th, the central abscess of the right cornea has increased in size. The ring-shaped infiltration in the left cornea is more marked, encircling a loss of substance in the centre, the bottom of which is perfectly black (Descemet's membrane lying bare). Diphtheritic deposits on lids disappearing. Profuse discharge of thin pus. Lids less swollen (disease passing into the blennorrhoic stage). The same treatment continued. April 18th, right eye, intense pyorrhœa; swelling of lids and conjunctiva moderate; whitish infiltration of upper half of cornea. Left eye, round, central perforation of cornea, about 4 *mm.* in diameter; small, regular pupil; iris and anterior surface of lens bare and perfectly clean; remainder of cornea, even the edges of the perforation, clear. April 19th, profuse discharge of thin pus; no chemosis. White infiltration of right cornea smaller. A white plug covers the central defect in the left cornea. April 20th, the white patches of both corneæ, a fibrinous-looking substance, contracting. April 21st, periphery of cornea clearing in both eyes. Discharge continues.

For the next ten days, under constant application of the iced compresses and cleansing of the eyes by irrigation, the inflammation varied in intensity. Then the ulcer in the right eye increased again, the greater part of the cornea sloughed, and the iris and lens were bulging. The peripheral two thirds of the left cornea cleared up, and the central ulcer contracted. On May 2d, however, the right eye began to swell and protruded; panophthalmitis set in, and, after its course, ended as usual in phthisis bulbi.

On May 8th, the left eye, which seemed to advance favorably toward recovery, began to swell likewise, protruded, was very painful, and, without sloughing of the cornea, passed through a moderately severe panophthalmitis, which, after two weeks' duration, left the cornea reduced in diameter, with total anterior synechia, and the eye somewhat shrunken and soft; perception of light lost.

June 17th, the patient was taken to the Institution for the Blind, in 34th Street and 9th Avenue, New York City.

Remarks.—Every one familiar with the clinical history of diphtheria of the conjunctiva, will recognize in the last case an example of the most dangerous form of this disease. If, in the first days of the diphtheritic inflammation, the cornea becomes hazy and infiltrated, the eye usually is lost, what-

ever the treatment may be. Such cases, fortunately, are not frequent.

I have no reason to consider the appearance of diphtheria to be accidental, for there was neither an epidemic of diphtheria in the city, nor was there a sporadic case under my care at the time when these patients were affected. The period of inflammatory swelling in both cases lasted only a day, then the characteristic deposit appeared, just as in the usual jequiritic ophthalmia, with the difference that, instead of being croupous, it was genuinely diphtheritic. The third—blenorrhoic—stage, completed the picture. True diphtheria from inoculation with jequirity has been described also by other observers. Dr. E. Grüning (see his instructive tabulation of twenty cases in the preceding number of these ARCHIVES, p. 158) states in ten of his cases, that dense membranes had formed *in the substance* of the palpebral and sometimes also *in the ocular* conjunctiva. The occurrence of diphtheria, and occasionally of diphtheria of the most deleterious kind, has therefore to be registered as one of the consequences of the application of jequirity infusion. In our cases we may inquire whether, on the one hand, it was contra-indicated by the nature of the preëxisting disease; and, on the other hand, whether the remedy was correctly prepared and applied or not.

To answer the first question, I may say that some objection may be raised, according to the rules and precautions laid down by De Wecker, as to the propriety of applying it in the first case, which was one of mixed granulations with a clear cornea. Yet these cases, as far as the conjunctiva is concerned, fall within the range of the indications of the remedy, for there was a chronic trachomatous condition of the conjunctiva, without any purulent or puriform discharge. The subsequent diphtheria was of a milder form and terminated favorably. The second case was clearly one of those in which the use of jequirity is considered indicated by De Wecker and every other writer on the subject. It was chronic follicular trachoma, without papillary or other swelling of the conjunctiva, and without discharge. The cornea was affected with old vascular pan-

nus, and sight reduced to $\frac{1}{200}$. Twice the application had been made in the left eye without great reaction; the third time, when both eyes were inoculated, an acute, most violent diphtheria, in the typical form of this disease, developed first in the eye inoculated for the first time, then in the other, and destroyed both eyes under my constant supervision at the hospital. No argumentation can mitigate the grief and mortification which this disastrous result left in my heart.

With regard to the second question, we have to inquire whether a three-per-cent. infusion, after a maceration of three or four hours, applied on the same day, is not too strong. Before it is decided whether the active principle of the infusion is a micro-organism, or a chemical agent, this question can be answered by practical experience alone. If the active principle is a chemical agent then dilution will weaken its efficacy proportionately, whereas a similar ratio could not be established with the same precision, if the jequirity ophthalmia depended upon the inoculation of micro-organisms. But even in this case a certain concentration of the inoculating fluid seems necessary to insure its action. On this point, as on that of the indications, further researches seem to be needed at present as much as ever before.

In spite of the unfavorable observations which I have communicated, and which are not the only ones on record, it is not my opinion that jequirity ought to be given up as too dangerous a remedy, but I think that its nature and action ought to be more carefully studied before we can *extensively* employ it for the human eye. No exception can, so far as present experience goes, be taken to its use in one group of cases, viz.: inveterate, dense pannus, with advanced cicatrization of the conjunctiva. It seems that in such cases the remedy is incapable of producing diphtheritic infiltration or purulent discharge to any great extent, for the cicatricial tissue, very scantily supplied with blood-vessels, cannot furnish nutritive material enough for either an infiltration into the depth of the conjunctiva, or for an abundant purulent secretion.

ON THE PATHOLOGICAL ANATOMY OF CONGENITAL COLOBOMA OF THE CHOROID AND IRIS.

By Dr. J. THALBERG, of St. Petersburg.

Translated by J. A. Spalding, M.D., Portland, Maine.

THE death of a child, æt. two years, from double croupous pneumonia gave me an opportunity to examine more closely two eyes, one of which exhibited a coloboma of the iris and choroid, while the other showed only a coloboma of the iris. The microscopic examination demonstrated, as will be seen further on, that there must have been an intra-uterine inflammation at the fœtal ocular fissure which interfered with the growth of the choroid and iris and caused a coloboma in these tissues. I have been unable to discover any thing in regard to the clinical history of the case. The child appeared somewhat wasted by the disease, but was otherwise well developed.

The post-mortem examination showed that the orbits and neighborhood were normal. The corneæ appear smaller than they really are, owing to a fold of conjunctiva having spread out over the outer third in the form of a cicatricial cord. The coloboma of the iris in each eye lies downward, and in the right eye somewhat inward. The enucleated left globe, owing to the extent of the coloboma in the choroid, appears as transparent as that of a white rabbit. The right eye is considerably smaller (microphthalmus), and has been pushed outward and upward by a partly solid and partly cyst-like tumor, which originates in the sclera near the optic nerve and extends to

the inner-lower angle of the orbit. The cavities into which the cyst is divided are filled with a redish fluid.

The left bulb, divided in the horizontal plane, measures 17 *mm.* in its antero-posterior diameter, and 15 *mm.* vertically. The lens is opaque at the posterior pole. The coloboma in the iris extends only to the ciliary body, which is intact. The coloboma of the choroid is heart-shaped, and at the equator of the eye is 8 *mm.* wide; it begins at the ora serrata, and terminates in a point at the optic nerve. The sclera is thickened over the whole extent of the coloboma. Near the optic nerve it is three times as thick as usual. The retina covers the entire extent of the coloboma, but near the margins of the latter, and close around the optic nerve, it exhibits numerous folds. It can be easily lifted away from the margin of the coloboma, but in the middle of the latter, where it is somewhat thinner, it adheres closely to the sclera. At one spot on the ora serrata the retina is raised above the choroid in the form of a cyst as large as the head of a pin. The choroid also, at the margin of the coloboma, is closely adherent to the sclera.

The right bulb measures 14 *mm.* by 15 *mm.* in diameter. The lens is also opaque at the posterior pole, and thicker than that in the left eye, while, owing to the minute size of the globe, it almost touches the optic papilla. The retina is thickened, folded, and partially detached about the optic papilla, but shows no other macroscopic alterations. The iris has a narrow slit, just like one made with the scissors. The sclera at the optic-nerve entrance is covered with a large tumor, partly cystic and partly cartilaginous, measuring 20 *mm.* in diameter.

Microscopic Examination.—The irides are profusely infiltrated with round cells, especially at the edge of the coloboma, where they are very much thickened. No especial endothelial membrane is visible. The proliferation of cells in the anterior layer of the iris has produced at the margin of the coloboma nodular and wall-like excrescences, which here and there surround the pigment situated along the edge of the coloboma. The pigment is much

darker than usual. The place of the pigment border is occasionally occupied by clusters of colorless round cells, which are in turn surrounded by irregular clumps of pigment. Beneath these, also, the tissue of the iris is extensively infiltrated with cells, while even here we see large and irregular masses of pigment.

The ciliary body in each eye, as well as the choroid of the right eye, is normal. The same may be said of the choroid of the left eye, as far as the edge of the coloboma. But from here to the optic-nerve entrance it is thickened by a deposit of numerous round cells, and suddenly terminates at the margin of the coloboma in the form of a ridge of closely aggregated, unpigmented, round and spindle-shaped cells; at the equator of the globe it assumes a structure which bears considerable resemblance to the sclera. It then is gradually lost in the coloboma in the form of scattered spindle-shaped, unpigmented, stroma cells which belong to the outer layers of the choroid, and of fibrous threads which contain a few scattered clumps of pigment. The cells and fibres are very closely adherent to the sclera at the margin of the coloboma. The margin of the coloboma is therefore partly composed of a thin layer of fibrous tissue appertaining to the external layers of the choroid, together with scattered stroma cells and clumps of pigment, and partly of a large number of regularly stratified layers of small round and spindle cells. The coloboma is devoid of blood-vessels; the chorio-capillaris disappears first. A membrane like Bowman's is found at intervals between the pigment epithelium and the sclera. Not even any rudiments of the choroid can be seen in the coloboma itself. The retina, therefore, lies here upon the inner layers of the sclera, and at the margin of the coloboma upon the rudimentary choroid which, as we have seen, is devoid of vessels.

The pigment epithelium of the retina increases in thickness just in front of the margin of the coloboma to a wall of black pigment, or is doubled over into the folds of the retina and forms a heap of large clumps of pigment, between which we see clusters of unpigmented round cells,

which probably have originated in a division of the nuclei of the pigment cells. From this wall, as far as the coloboma, the pigment epithelium again exhibits a layer of regular but faintly pigmented, or alternately pigmented and unpigmented cells. The wall can be followed a short distance upon the coloboma in the form of a simple layer of unpigmented cells. It then subdivides into a complex layer of unpigmented and generally smaller cells with oval nuclei, which, after forming a ridge-like swelling, either suddenly cease, or, by further subdivision, send out round cells resembling lymph corpuscles, which, since the *memb. limitans ext.* and the rods and cones are here entirely absent, unite with the granules of the external granular layer. Moreover, since numerous cells have made their appearance even in the inner layers of the retina, their confluence with the cells of the pigment epithelium, resp. the granules of the retina, produces a moderately close union between the retina and the sclera.

The retina is chiefly altered at the entrance of the optic nerve and in the coloboma of the choroid, where it is greatly thickened. In the middle of the coloboma it suddenly grows thin, and becomes a narrow strip (raphe). The thickening is chiefly due to proliferation of the granules of both granular layers—especially the external—but additionally by hypertrophy of the connective-tissue portion of the layers of nerve fibres and ganglion cells. The proliferation about the optic nerve and at the margin of the coloboma has caused the retina to appear twice or three times as thick as normal, so that at the places where this tissue has folded the inner and outer granulated layers as well as both granular layers have been so extensively pushed through one another, that of the inner granulated layer only a few patches are visible, surrounded by proliferated granules, while the external granulated layer from the action excited by the proliferation of granules has been reduced to a few fine fibres of connective tissue, or pushed out into a wide-meshed network of fibres of similar structure. But where the retina has escaped this folding process, the two granulated layers are unaltered, al-

though the granular layer, the layer of nerve fibres, and the layer of ganglion cells appear excessively thickened. In both of the latter the nervous elements have been destroyed, a few cells and nuclei alone remaining. The supporting fibres, especially, appear like thick columns in the foreground, and can be distinctly traced throughout all the layers of the retina. The layer of rods and cones has likewise disappeared. Behind the *memb. limit. ext.* and the pigment epithelium there lies a single layer of a finely granular mass, containing a few round cells. A similar mass has lifted the retina from the choroid in a cyst-like protuberance near the ora serrata. The *memb. limitans ext.* has been destroyed by the extensive proliferation of granules at the margin of the coloboma, so that the granules of the external granular layer now rest directly upon the sclera. The retina in the middle of the coloboma suddenly grows extremely thin, and from its rudimentary development appears to have undergone atrophy. Its various layers can no longer be distinguished. It resembles a thin membrane composed of fine fibres of connective tissue with a few nuclei scattered about. We can occasionally get a glimpse of the inner granular layer. The raphe does not possess any blood-vessels, nor does the adjacent thickened portion of the retina, but they abound in the rest of this tissue. The retina is here closely united with the sclera by anastomosis of its cells, nuclei, and connective-tissue fibres with those of the corresponding portion of the sclera.

The retina of the right eye has degenerated into fibrous tissue only close around the optic-nerve entrance, while it has at the same time lost its nervous and percipient elements like that of the left eye. The proliferation of the cells and hypertrophy of the connective-tissue elements have also caused a thickening of the retina distinctly visible to the naked eye. Toward the ora serrata, the retina appears atrophic; the granules have not undergone proliferation, but the nerve fibres, ganglia, and rods and cones have disappeared; the supporting fibres alone are visible. But none of the alterations suggest a former fissure in the retina, for the latter is everywhere entirely closed over, while its pigment epithelium is normal and intact.

The optic nerves simply exhibit a fibrous connective tissue, with numerous cells and nuclei: nerve fibres are absent. The opacity at the posterior pole of both lenses is composed of a conglomeration of lenticular fibres which have degenerated into a finely granular mass. The opacity is surrounded with lenticular fibres which have degenerated into swollen round or oval hyaline masses, and fibres which resemble connective tissue. Cross-sections exhibit a very irregular mosaic of more or less swollen hexagonal lenticular fibres. In the anterior portion of the lens, in close proximity to the anterior capsule, we notice between the unaltered lenticular fibres numerous fissures which are filled with a finely granular mass and a few lenticular fibres which have simply swollen or degenerated into a row of ninepin-shaped pieces. Still further in front, the nuclei of the lenticular fibres have enlarged and split into two.

The interior portion of the sclera appertaining to the coloboma in the choroid exhibits slight proliferation of cells and nuclei. But at the optic-nerve entrance the infiltration with cells becomes so noticeable that the bundles of fibres are pressed apart by connective tissue, rich in cells, and the sclera thereby rendered twice or three times as thick as usual. The proliferation of connective tissue in the sclera, at the optic nerve of the right eye, however, has been so extensive as to produce a tumor-like hypertrophy with the formation of a cyst. The coarse portion of the tumor resembles the sclera in structure, whilst the cyst-wall is composed of thick concentric layers of connective tissue, enveloped on the interior with a compound layer of round, epithelial-like cells.

The anatomical examination of these eyes shows, in concordance with Deutschmann,[1] that an intra-uterine inflammatory process at the fœtal ocular fissure produced so extensive a degeneration of the retina, together with the formation of a coloboma of the choroid and iris, as well as hypertrophy of the sclera by proliferation of the connective tissue surrounding its fibres, that finally in one eye there arose a cyst-like tumor with defective development of the globe.

[1] *Klin. Monatsbl.*, Marz, 1881.

The inflammatory alterations had further excited so well marked disturbances in the nutrition of the eye that the lenticular fibres at the posterior pole become swollen, underwent degenerative changes, and even became partially reabsorbed. Rumschewitsch,[1] on the contrary, is of the opinion that the rudimentary development is not due to fœtal inflammatory processes, but to entirely different circumstances. It is indeed possible that other causes may have been active in other cases, but in our case an inflammation with proliferation of cells must have been the cause of the fissure.

The most important pathological alterations dependent upon proliferation of cells are to be found in the sclera, choroid, and especially in the retina near the entrance of the optic nerve. The inflammation, therefore, appears to have started from this same point. And further, since the pathological alterations corresponding to the former retinal fissure are still present, the inflammation must have ensued at a time when the fissure had not yet closed over. While in most of the other cases hitherto reported (Manz[2]) the retina was absent in the coloboma, in our case the inflammatory process has neither checked the formation of its individual layers, with exception of the pigment epithelium, nor has the proliferation of the cells of the pigment epithelium and of the granular layers checked the closure of the retina. A partial closure of the fœtal retinal fissure, however, appears to have been affected by means of a fine raphe of fibrous nucleated connective-tissue, with small islands corresponding to the inner granulated layer. Beyond this the proliferation in the retina has destroyed the nerves and percipient elements, and finally produced a transformation of the already developed pigment epithelium at the margin of the coloboma into a heap of unpigmented round cells and clusters of pigment, or interfered with its normal development in such a manner that it amounts to nothing more than an imperfect pigmentation of the cells or their dissolution into small round cells, which, forming a ridge-

[1] *Centrlbl. f. Aug.*, Mai, 1882, p. 141.

[2] *Græfe-Sæmisch*, Band ii., Theil 1, page 83.

like swelling, either suddenly cease at the margin of the coloboma, or gradually coalesce into the granules of the external granular layer, with absence of the rods, cones, and *memb. lim. intern.* The dissolution of the pigment cells into small round cells at the edge of the coloboma is sufficient to account for their absence in that part of the retina which covers the coloboma.

It is well known that we can only differentiate between the sclera and choroid when the fœtal ocular fissure has finally closed over (Manz[1]). But since in our case the inflammatory process precedes this condition, continues during the closure of the retina, and leads to a union of the proliferating cells of the pigment epithelium and of the external granular with the underlying membrane, which at present cannot be differentiated into sclera and choroid, so in a similar manner does the same process influence the development of that portion of the choroid which corresponds to the retinal fissure, either leaving it rudimentary, or even preventing it entirely by giving rise to a production of round cells. For while the choroid everywhere else is normally developed, these round cells appear more and more abundantly toward the margin of the coloboma, so that the choroid either ceases here entirely in the form of a ridge of round cells, or gradually becoming thinner, it exhibits its rudimentary development in the form of stroma cells and fibres, together with scattered clumps of pigment, as we have already mentioned. The chorio-capillaris is entirely absent at the margin of the coloboma. The degenerated retina at this place consequently lies either upon the rudimentary choroid, but in the coloboma itself, upon a structureless membrane like that of Bowman's, or the inner layers of the sclera infiltrated with cells and nuclei. The coloboma of the choroid must, therefore, be regarded as an arrest of development produced by an inflammatory process upon the fœtal retinal fissure while it was about to close over.

In the other eye the inflammatory process did not interfere with the total closure of the retina or normal develop-

[1] *L. c.*, page 82.

ment of the choroid, appearing to confine itself exclusively to the place of entrance of the optic nerve, where it caused extensive thickening and degeneration of the retina. It seems, however, to have hindered the development of the iris, and produced a fissure in this tissue just as in the other eye, though not quite so extensive. The margins of the coloboma of the iris exhibit about the same results of the inflammatory process as we have already noticed at the margin of the coloboma of the choroid.

The extensive degeneration of the retina around the optic nerve, the atrophy of the optic nerve, the destruction of the nervous and percipient elements in the entire retina, together with the destruction of the lenticular fibres, show that the child could not have possessed any vision.

It is by no means improbable that the appearance of the same intra-uterine inflammation at the fœtal ocular fissure in a still earlier stage of development, or in greater intensity, may lead to that form of anophthalmus in which we discover simply a rudimentary globe, or even none at all, as is by no means extremely rare amongst the laboring classes. Thus, amongst 1,296 children affected with eye-dis eases at the St. Petersburg Elizabeth Hospital in 1881, there were five with double anophthalmus, and one of unilateral anophthalmus and microphthalmus, with a coloboma in the other eye. The only reason why the eye appears to have remained *in situ* behind the conjunctiva seems to depend upon the presence of the conjunctiva in the form of a broad band which covers the external third of both corneæ, while the assertion that a more extensive inflammation may cause anophthalmus is favored by the case just cited, in which the same inflammatory process caused anophthalmus on one side and microphthalmus with formation of coloboma on the other. Manz[1] speaks as follows: "When we consider to what degree of destruction the phthisis, no matter how caused, of any perfectly developed eyeball may lead, so that finally it is only the coarse sclera which prevents it from total annihilation, it is plain that even total

[1] *L. c.*, page 124.

destruction might similarly ensue to an eye that was not yet perfectly developed."

It is plain, at all events, that an eyeball must have existed for some time for an orbit to form about it, the skin to fold into lids, and a conjunctiva to surround it in its entirety.

A CASE OF CONGENITAL FISTULA SACCI LACHRYMALIS.

By WHEELOCK RIDER, of Rochester, N. Y.

AN examination of ophthalmological literature reveals but few reported cases of congenital fistula of the lachrymal organs. Steinheim [*Klin. Monatsbl. f. Aug.*, xiii., p. 303] describes a fistula of the duct, opening upon the surface of the upper lid, believed to have existed at birth. Terson [*Presse Méd. Belg.*, 1873, p. 227] reports two cases of fistula of the sac, but whether these were congenital is doubtful. Manz [Graefe-Saemisch: "Handb. d. ges. Aug.," ii., p. 113] writes: "It is still doubtful whether a congenital fistula of the lachrymal sac has been observed, though the possibility of its appearance, as an occasional failure of the nasal duct to close, is not to be denied. In this case it should be viewed as an arrest of development." The following case may therefore be of interest.

W. R. æt. 21. About one half inch below the inner canthus of the right eye, and over the course of the lachrymal sac, appears the opening of a canal, sufficiently large, to admit for a short distance a No. 8 Bowman's probe. The integuments about the opening, and the walls of the canal, are slightly indurated, thus showing on pressure the general direction, viz.: upward and inward. The attempt to pass a probe through into the sac has never been successful, the canal being contracted somewhere in the course. By closing the nostrils and forcing air into the nose, tears collected in the sac may be driven through the fistula upon the cheek; no discharge taking place however spontaneously. The eye is in all other respects normal.

This fistula is without doubt congenital, it being noticed when the individual was but a few days old. Being a source of no discomfort operative interference is not advised.

AN UNUSUAL TOLERANCE OF A FOREIGN BODY IN THE FUNDUS OF THE EYE.

By WHEELOCK RIDER, of Rochester, N. Y.

DR. H. KNAPP (these ARCHIVES, xi., p. 222) has reviewed twelve reported cases of penetrating injuries of the eye, where foreign bodies, lodged in various places in the fundus, have remained for a considerable period without serious irritation being produced. To these cases he has added one which came under his own observation. The following may be added to the cases mentioned above.

Mr. B., student, was struck in the left eye with a piece of steel from a chisel while opening a box, on March 15, 1884. Examining the eye shortly after the accident, he found the ocular conjunctiva slightly injected, especially near the inner margin of the cornea. Suffering no pain, and thinking the particle had been brushed away by the lids, he consulted no one until six weeks after the injury. He had now noticed a blur in the visual field, below and to the right side. The eye appeared free from irritation. No external scar was visible. By ophthalmoscopic examination all parts appeared healthy and uninjured with this exception: above the disc, at a distance of about three times its diameter, the fragment of steel could plainly be seen, its freshly fractured surface glittering brightly. It was roughly triangular in outline, one portion imbedded in the retina, choroid, and sclerotic, the other projecting forward into the vitreous. There was a very slight discoloration about the point where it was fixed, and a little opacity could be distinguished in the vitreous before it.

In this case the foreign body had passed through the sclerotic, leaving the lens and iris uninjured, and thus the eye was in excellent condition for examination. Ten weeks after the injury the patient reported the eye feeling better than at any time since the accident.

APPENDIX TO THE PAPER ON BLINDNESS FROM FACIAL ERYSIPELAS.

PAGE 83 OF THIS VOLUME.

By H. Knapp.

In the April number (1884) of *Zehender's klin. Mon.*, Dr. A. Carl, of Frankfort o. m., publishes a case of blindness from erysipelas, illustrated by two colored drawings. The observation is not so complete as that reported by myself, but shows that in both cases the picture of the disease must have been identical, with the exception of the retinal hemorrhages in ours, which, thus far, have not yet been noticed in any other. The following, an abstract of Dr. Carl's observation, may, as the thirty-seventh case, be added to the bibliography at the end of my paper.

(37) *August Carl*, on the special etiology of the optic-nerve atrophy following facial erysipelas, *Zeh. klin. Mon.*, 1884, p. 113. The erysipelas began at the tip of the nose. First the lids of the left, then those of the right, eye swelled. On the tenth day pus escaped from the left nostril and from an abscess at the inner canthus of the left eye. No complaint of sight. On the fifteenth day, at the patient's admission to the hospital, Dr. Harbordt found total amaurosis, protrusion, and impeded mobility of the *right* eye; besides, visible thrombosis of the supra-orbital and frontal blood-vessels, which, in the shape of a solid cord, passed over the forehead. In the right temporal and parietal regions which had been invaded by the erysipelas several abscesses formed. The thrombosis gradually disappeared.

Dr. Carl saw the patient for the first time six or seven weeks after the occurrence of blindness in the right eye. He still found

the eye slightly protruded; the media clear, the pupil medium-sized, the vision extinct. The left eye, which always had been the weaker, had S = $\frac{1}{5}$, but showed nothing notable. In the right eye the optic disk was well defined, not swollen, but milky opaque, the same as the surrounding zone of the retina (fig. 1). The retinal vessels were reduced in number, partially filled with blood, partially converted into white cords. One artery and two veins corresponding to it were normal.

Two weeks previously Dr. Krüger had examined the patient with the ophthalmoscope, and entered the following note in his case-book: "Arteries of disk and retina completely empty of blood; veins filled only in parts (streckenweise); retina white-gray.

Four months after blindness had set in, the optic disk was totally atrophic; twenty months later it was white as chalk. The two above-mentioned veins, were then still not materially changed; the artery, however, had disappeared. Some very thin, short vessels were on the disk and its nearest vicinity. Three white cords, one of them very broad, passed from the disk some distance through the retina, and terminated abruptly. In the region of the yellow spot there were some grayish-black patches, some white dots, and a white, interrupted cord, evidently the remnant of a blood-vessel (drawing 2).

Dr. Carl considers the suspension of the retinal circulation, not neuro-retinitic, as the cause of the blindness and the subsequent intra-ocular changes.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FIRST QUARTER OF THE YEAR 1883.*

By H. MAGNUS, Breslau; C. HORSTMANN, Berlin; and A. NIEDEN, Bochum.

WITH THE COÖPERATION OF

C. E. Fitzgerald and Ferguson, Dublin; E. Marckwort and P. von Mittelstädt, Antwerp; Dantone, Rome; Hirschmann, Charkow; S. M. Burnett, Washington; Schjötz and Ole Bull, Christiania, etc.

Translated by Dr. F. E. D'Oench, New York.

C.—IRIS AND UVEAL TRACT, GLAUCOMA, SYMPATHETIC OPHTHALMIA, REFRACTIVE MEDIA (LENS AND VITREOUS BODY), RETINA AND FUNCTIONAL DISTURBANCES, OPTIC NERVE, INJURIES, FOREIGN BODIES (PARASITES), OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

By Dr. A. NIEDEN.

I. *a.*—IRIS.

210. Borthen-Lyder. Aniridia and aphakia. *Zeh. klin. Monatsschr. f. Augenhk.*, vol. xxi., p. 62.

211. Galezowski-Daguenet. Maladies de l'iris. Diagnostique et traitement des affections oculaires.

212. Gayet. Formes rares de traumatisme amenant tumeur

* Continued from p. 527, vol. xii.

de l'iris faisant hernie sous la plaie cornéenne. *Soc. franç. d'ophth.* 1880.

213. RAMPOLDI. Una nuova causa di midriasi. *Ann. d'ottalm.*, vol. xi., 6.

GALEZOWSKI (211) subdivides iritis according to its causes into the following groups: syphilitic, rheumatic, gouty, and blennorrhoic. He describes the symptoms of each in detail and discusses particularly the differential diagnosis.

BORTHEN (210) saw the iris and lens completely torn from the left eye of a man in consequence of a blow with a piece of wood, while the other parts had remained relatively uninjured. The eye had been short-sighted; later a choroidal coloboma surrounding the papilla, and a circumscribed choroidal atrophy on its temporal side outward of it were discovered. $V < \frac{20}{200}$, not improved by glasses or a diaphragm. Epicritical discussion of the case.

GAYET (212) observed two cases of hernia of the iris after an injury and reports one of them. In explaining them Abadie and Armaignac have recourse to the experiments of Masse on animals (v. *Arch. f. Augenheilk.*, vol. xi., rep. Nos. 828, 1261, 1262). According to Gayet the tumors of Masse are not cysts but the results of epithelial transplantation. MARCKWORT.

Three years ago RAMPOLDI (213) observed marked mydriasis in a young girl of nineteen, after probing her naso-lachrymal canal a few times. It gradually disappeared when the probing was discontinued, but at once returned when probing was renewed. Since then Rampoldi carefully observed the pupils of his patients on whom he had operated for strictures, and noticed the same peculiar phenomenon in nine other cases: in two of them the pupil was very much dilated and immovable; in the others it reacted, but was wider than that of the other eye. In one case the first introduction of the probe produced, besides mydriasis, an epileptiform attack in a young woman, who never had any before. The power of accommodation was not affected in the cases in which it was tested. Rampoldi ascribes the phenomenon to irritation of branches of the sympathetic nerve, produced by the probe. (The reviewer can confirm these observations by his own experience.) DANTONE.

I. *b.*—CHOROID.

214. BOCK, E. (Vienna). The miliary tubercles of the uvea. *Virchow's Arch.*, xci., p. 434.

215. Bock, E. A sarcomatous tumor of the choroid containing biliverdin. · *Virchow's Arch.*, xci., p. 442.

216. Chelkowsky. Continuous compression of the blood-vessels of the neck the cause of recurrent irido-choroiditis. *Oesterr. Monatsschr. f. Thierheilk.*, vol. viii., 3.

217. Courserant. De la chorioidite antérieure ; leçon recueillie par le Dr. Leviste. *Gaz. des hôp.*, Mar., 1883.

218. Dimmer, F. (v. Arlt's clinic). Case of metastatic choroiditis after extraction of a molar tooth. *Wiener med. Wochenschr.*, 1883, No. 9, p. 241.

219. Fontan. Ossification de la chorioide. Examen de trois bulbes oculaires atteints de cette ossification. *Rec. d'ophth.*, Apr., 1883.

220. Gayet. Coincidence de chorio-rétinite double et d'anciennes lésions ganglionaires. *Ann. d'ocul.*, Mar.–Ap'l, 1883.

221. Goldzieher, W. Exudation in the supra-choroid, due to external choroiditis. *C. f. A.*, vol. vii., p. 41.

222. Schöler. Case of blindness due to metastatic sarcomata of the choroid in both eyes. *Berl. klin. Wochenschr.*, No. 7, 1883.

According to Courserant (217) it is necessary to dilate the pupil widely with atropine in order to make a thorough examination in *anterior choroiditis*. This disease is frequently connected with affections of the uterus. Marckwort.

Fontan (219) describes three cases of *formation of bone tissue* in the choroid ; it resembles the bone tissue of the cranium. Marckwort.

Goldzieher (221) refers to a case reported by Brailey (*v.* these Arch., vol. xii., p. 134), in which *a dense layer of connective tissue* was found *between* the *choroid* and *lamina fusca*, interspersed with broad cystoid lacunæ. The author has described them before this, and shown them to be the result of an inflammatory process, especially in globes which had been enucleated to prevent sympathetic ophthalmia. This inflammation of the supra-choroid is the anatomical basis of serous choroiditis which leads to diffuse cloudiness of the vitreous. As a result of this external choroiditis the supra-choroid is converted into a delicate sinewy membrane, which gradually changes into dense fibrous tissue, and thus produces those ominous symptoms of irritation which may give rise to sympathetic ophthalmia in the other eye.

GAYET (220) describes a peculiar etiology of a case of chorio-retinitis of both eyes. The right hand of a girl of nineteen was injured, which was followed by inflammation of the glands of the right side; six months later the eyes became affected, the left first, then the right. Gayet tries to establish a connection between the injury of the hand and the affection of the eyes, and says that the case "shows a series of changes, commencing with an injury of the hand, passing on through the cervical ganglia, reaching the left eye, then the right, and returning to the lymphatic system by way of the right preauricular ganglion."(!) MARCKWORT.

BOCK (214) found symptoms of tuberculosis in 19 globes out of 23—82.7 %—in persons who had died of that disease. The number of tubercles varied between 2 and 49. The largest tubercles were generally found around the papilla, but also around the vortices and æquator bulbi. He sometimes observed hemorrhages in tubercles of medium size. There was almost always diffuse choroiditis, which sometimes extended along the sheaths of the nerves and blood-vessels into the sclera. The pigment was only slightly affected, being somewhat paler near the tubercles. The retina was œdematous. The inflammation had also affected the ciliary body and choroid in 43.5 %; but no tubercle was ever found in the ciliary body, and only one in the iris, therefore in 4.4 %.

BOCK (215) also observed in the case of a man of forty affected with a *tumor* of the liver and sarcomatous dyscrasia, that the left eye was amaurotic and contained a tumor springing from the *choroid.* It was composed of polygonal or cylindrical nucleated cells which were very similar to the cells of the liver. The structure of the neoplasm very closely resembled an adenoma of the liver, a resemblance which was increased by the peculiar rosette-like arrangement of the cells and the presence of drops of bile in the centre of the rosettes. Chemical analysis showed the presence of a considerable quantity of biliverdin. (*V.* the case of BEHRING-WICHERKIEWICZ, Rep., *Arch. f. Augenheilk.*, vol. xii., 2, p. 252.)

DIMMER (218) observed the development of pyæmia in a boy of thirteen, produced by an injury of the left lower jaw during the extraction of a molar. The injury was followed four weeks later by *metastatic choroiditis* of the *right eye*, ending in atrophy of the globe.

SCHÖLER (222) reports a case of *metastatic carcinoma of the*

choroid in both eyes, observed in a woman of thirty-three. In the right eye, which was first affected, the retina was extensively detached, the papilla swollen, the retinal blood-vessels dilated; around the disk there was a grayish-white belt, the papilla itself containing some opaque epithelial nests. In the retina of the left eye there were only in the region of the macula lutea numerous grayish-white dots, the size of a pin-head or smaller, without any pigment, and some with a slightly reddish tinge. The patient was suffering from a carcinoma of the mamma, which had been extirpated as far as possible, but had produced metastases. Without doubt the infiltrations of the fundus were of a metastatic nature. They rapidly developed during the ten months they had been observed, while the detachment of the retina in the left eye disappeared spontaneously, the swelling of the papilla subsided, passing into atrophy, while in the right glaucoma set in, without, however, leading to perforation of the sclera. Probably this was a case of carcinoma of the capillaries of the short posterior ciliary arteries, gradually developing into a carcinomatous infiltration of the whole choroid without any marked excrescences.

II.—GLAUCOMA.

223. CHIBRET. Nouveau traitement chirurgical du glaucome. Thérapie de l'affection. *Soc. franç. d'ophth.*, 1883.

224. GALEZOWSKI. Du glaucome sympathique. *Rec. d'ophth.*, Apr., 1883.

225. HOCQUARD, E. and MASON, ALB. Microphthalmie compliquée de glaucome chronique. *Arch. d'ophth.*, vol. iii., No. 3.

226. SULZER, DAVID. Iridectomy in glaucoma. Zurich, 1882.

GALEZOWSKI (224) believes that any one of the membranous coats of the eye can be affected with sympathetic ophthalmia; the existence of *sympathetic glaucoma* cannot be doubted, as a case recently observed shows. A woman of forty-one, whose right eye had been injured three years before and had become atrophic, complained of her left eye. Conjunctiva: large swollen veins, pericorneal capillary injection; cornea clear; iris of almost normal color, but pupil not reacting upon light; some posterior synechiæ at the lower margin; deposits of pigment upon the anterior capsule; media hazy, but disk visible; marked excavation; slight pulsation of the artery; V = 1, + J 1. Visual field considerably

contracted downward and outward. The majority of these symptoms disappeared after enucleation of the right eye. As the pericorneal injection and increased tension remained, iridectomy was performed. The contraction of the visual field was not materially affected. (No evidence that the glaucoma was sympathetic, Ed.)

Galezowski calls particular attention to the masses of pigment upon the anterior capsule ; he considers this simultaneous occurrence of iritis and glaucoma in the same eye—at least in this case —as characteristic of sympathetic glaucoma. MARCKWORT.

HOCQUARD (225) enucleated the left eye, blind from chronic glaucoma, in a woman of thirty, with microphthalmus of both eyes, and describes its microscopic appearance : globe reduced in all its dimensions, but not otherwise deformed ; lens of normal size, anterior chamber shallow, Fontana's space closed by peripheric adhesion of the iris, which, at this point, is pushed forward by the ciliary processes, sclerosed and partly united with it ; choroid and retina detached, the former slightly, the latter considerably, changed in its structure ; disk excavated. The author explains the case according to the theory of Knies-Weber : closure of Fontana's space by the iris being pressed forward and swelling of the ciliary processes due to disturbances of the circulation in the microphthalmic globe. Sympathetic irritation was transmitted to the other eye by the ciliary nerves, in which inflammatory proliferation of connective tissue was observed.

V. MITTELSTÄDT.

CHIBRET (223) describes a *new method of operating for chronic glaucoma :* An oblique incision is made through the lamellæ of the cornea, 2-3 *mm.* from the margin, and the wound opened daily for three weeks, so that the aqueous escapes. If the iris falls into the wound and does not go back of its own accord, eserine is instilled. Report of two cases, in which massage was employed besides. Poncet thinks that in Chibret's method Descemet's membrane falls into the wound and thus prevents its firm closure in a favorable manner. A general discussion of the treatment of glaucoma followed the reading of Chibret's paper; Courserant spoke against repeated paracentesis and Gayet against Chibret's method. Panas and Masselon saw favorable results from cicatrisotomy. Abadie performs iridectomy in acute glaucoma, and if this fails, sclerotomy. In one case Abadie stretched the nerve according to Badal with good result (*v. Ann. d'ocul.*, 1882, Nov. and Dec., and rep. in *Arch. f. Augenheilk.*, vol. xii.,

3, p. 371). Meyer and Galezowski recommend myotics when operative treatment fails; of these Galezowski places pilocarpine far above eserine. MARCKWORT.

SULZER (226) reports the result of the glaucomas operated during the last twenty years at Horner's clinic, with notes as to the final result. Of 103 cases of simple glaucoma 22.67 % were improved, in 37 % the condition remained unchanged, in 23 % sight slightly diminished but was still useful, in 3.8 % vision at once decreased, and in 13.6 % it deteriorated afterward.

In 149 cases of inflammatory glaucoma 72.5 % were improved; in 11.3 % serviceable sight was preserved, in 10.1 % a part of it. In 4.08 % vision was impaired at once, in 2.02 % later. These numbers clearly demonstrate the usefulness of Graefe's operation.

III.—SYMPATHETIC OPHTHALMIA.

227. BRENNER, E. Case of sympathetic ophthalmia after diffuse tuberculous inflammation of the whole uveal tract in the primarily affected eye. Inaug. Dissert., Königsberg, Jan. 22, 1883.

228. GUTMANN (Hirschberg's clinic). Case of sympathetic ophthalmia. *C. f. A.*, vol. vii., p. 53.

229. PONCET. Anatomie pathologique d'un moignon d'œil ayant subi l'amputation de Critchett et determiné une ophthalmie sympathique. *Soc. franç. d'ophth.*, 1883.

230. SEBATERIE. De l'amputation du segment antérieur de l'œil comme traitement des accidents sympathiques. *Gaz. d'ophth.*, March, 1883.

231. BENSON. Frequency of papillitis in sympathetic ophthalmitis.

Mr. BENSON (231) notes the rarity of any account of the condition of the disk in the sympathizing eye in the recorded cases of sympathetic ophthalmia, whereas in the descriptions of the pathological condition of the exciting eye neuro-retinitis or papillitis is constantly observed. If then it be allowed that the sympathetic inflammation may be conveyed through the optic nerve and commissure, why may not papillitis be one of the earliest signs in the sympathizing eye? B. suggests that it is probable that most cases of sympathetic inflammation are ushered in by a more or less marked papillitis, and that it is advisable in all cases where sympathetic inflammation is feared to examine systematically, and at

short intervals, the papilla of each eye and to note its condition. (*Oph. Rev.*, vol. ii., May, 1883, p. 136.) FITZGERALD.

BRENNER (227) observed an adherent leucoma and *diffuse irido-cyclitis* in the left eye of a girl of eleven, while in the right there was *serous iritis* and *occlusion of the pupil.* He assumed the existence of sympathetic ophthalmia, enucleated the left eye and began inunction, whereupon the exudation disappeared. Microscopic examination revealed the symptoms of general tuberculous affection of the uveal tract, giant-cells, etc. The clinical history does not clearly prove the existence of sympathetic ophthalmia.

GUTMANN'S (228) case was one of a boy nine years old, in whom sympathetic ophthalmia developed in the other eye $2\frac{1}{2}$ years after the injury of the first, producing total occlusion of the pupil and cyclitic membranes. Various operations (Critchett, Wenzel, etc.) were tried without success, but finally a central pupil was obtained by iridotomectomy, with $V = \frac{1^{5}}{2\,0\,0}$, a good result for the present.

PONCET (229) reports a case of sympathetic ophthalmia due to the amputation of a stump according to Critchett. Poncet thinks the irritation was transmitted through the ciliary nerves.

MARCKWORT.

SEBATERIE (230) attended the ophthalmic clinic of Dr. Gillet de Grandmont, and believes that the experience gained by him in this time entitles him to recommend amputation of the anterior segment of the globe instead of enucleation. MARCKWORT.

IV.—REFRACTIVE MEDIA.

a.—LENS.

232. ABADIE. Accidents immédiats consécutifs â l'extraction des cataractes. *Soc. franç. d'ophth.*, 1883.

233. ARMAIGNAC. De l'opération de la cataracte chez les diathésiques et les cachectiques. *Rev. clin. d'ocul. du sud-ouest*, December, 1883. No failure in a number of extractions complicated with constitutional diseases. MARCKWORT.

234. CARRÉ. Cataracte sénile. Manuel opératoir de l'extraction. *Gaz. d'ophth.*, 1883, Nos. 1 and 2. Advises iridectomy.

235. CHAVERNAC. Extraction de la cataracte retour à la méthode de Daviel. *Ann. d'ocul.*, Jan., Feb., 1883.

236. HIRSCHLER J. Erythropsia in aphakia. *Wien. med. Wochenschr.*, 1883, 4–6.

237. GALEZOWSKI. De la nouvelle méthode de l'extraction de la cataracte sans excision de l'iris. *Rec. d'ophth.*, Feb., 1883. *Cfr.* discussion on extraction of cataract in the French Ophth. Soc. and

238. GALEZOWSKI. A new method of cataract extraction without excision of the iris. *The Lancet*, Jan. 20, 1883, p. 92.

239. JEAFFRESON. A new operation for cataract. *The Lancet*, Jan. 27, 1883, p. 167.

240. MARION V. KARWAT. Contributions to the affections of the eye in atheroma of the carotid (Prof. Michel). Inaug. Diss., Würzburg, 1883.

241. PANAS. Cataracte traumatique. *Journ. de méd et de chir. path.*, Feb., 1883.

242. SMITH. Growth of crystalline lens. *Trans. Ophth. Soc. of Unit. Kingd.*, iii., 1883, p. 79.

243. TERSON. Des moyens d'éviter la suppuration de la plaie dans l'extraction de la cataracte dans le cas de catarrhe du sac lachrymal. *Soc. franc. d'ophth.*, 1883.

KARWAT (240) reports further observations made at Würzburg on the influence of **atheroma of the carotid** on the development of **cataract.** 1. Four cases of unilateral cataract in atheroma of only one side or at least more marked on the side on which the cataract had developed; two of the patients are between the ages of fourteen and sixteen, the other two between fifty-eight and sixty-four; three of the cataracts belonged to the right, one to the left, eye. 2. Twenty cases of cataract on both eyes, which were more advanced and developed earlier on the side on which the atheroma was more marked; eleven were men, nine women, the average age sixty-one. In eight cases the cataract was more advanced on the right, in eleven on the left; once both lenses were equally cataractous, while the atheroma was more marked on one side than the other. 3. Two cases of cataract of both eyes, in which both the cataracts and the atheroma were equally developed on both sides. 4. Cataract in atheroma of the carotid complicated with struma. Two cases of embolism of the central retinal artery are also reported which were probably due to atheroma of the carotid.

HIRSCHLER'S (236) observation of *erythropsia in aphakia* is of great value as it was made upon himself, his left eye having been operated for cataract, with a broad coloboma. Erythropsia

began every evening, five months after the operation, lasted about an hour and then gradually disappeared without affecting the vision. It could be suppressed by diminishing the palpebral aperture, and may therefore be ascribed to irritation of the peripheric part of the retina, the broad coloboma permitting light to strike it at a certain angle.

PANAS (241) discusses two kinds of traumatic cataract: one is due to direct injury of the capsule, the other to a disturbance of the nutrition of the choroid or ciliary processes by the injury; the latter kind develops very slowly and is much more common than is generally supposed. MARCKWORT.

GALEZOWSKI (237 and 238) has modified the usual method of extraction, and performs an iridectomy only in exceptional cases, for which he gives the following reasons. Excision of the iris does not prevent inflammation, and frequently is the cause of secondary cataracts (?); the scleral and sclero-cornea. wounds endanger the final result much more than corneal wounds, and never prevent severe inflammation of the iris and cornea. Galezowski now enters Gräfe's knife at the sclero-corneal margin, 3 *mm.* above the horizontal diameter, and makes the counter-puncture at the corresponding point opposite; the section lies in the cornea, its apex 2 *mm.* from the margin. He performs cystotomy with the knife immediately after entering it, before making the counterpuncture. He excises the iris only (1) when the iris falls before the knife in making the section; (2) in traumatic cataracts with posterior synechiæ; (3) when the lens in escaping is entirely covered with iris; (4) when the iris is torn by the escape of a hard, large cataract. Results: In seventy-four cases three of slight iritis, one severe iritis, and two iris-herniæ. MARCKWORT.

Three papers read at the Soc. franç d'ophth., on cataract-extractions, were followed by a discussion in which Panas and Abadie declared in favor of spray, Wecker and Courserant against it. Meyer does not use the spray during the operation, but just before it, in order to disinfect the air. Meyer generally does not change the bandage before the second or third day; Wecker and Panas change it within the first two days without opening the lids. Panas and Meyer think a large wound suppurates more easily than a small one, while Abadie considers the size of the wound without influence in this respect. Meyer objects to Galezowski's

method of operating without an iridectomy, because it requires a larger section, which Galezowski denies. Wecker ascribes the cause of suppuration to the wound in the capsule, which Abadie also thinks plausible. MARCKWORT.

JEAFFRESON (239) is also opposed to Galezowski's method, which is not new, as it has been described by Renin.

FERGUSON.

CHAVERNAC (235) recommends *Daviel's old method of extraction*, a flap-section downward without an iridectomy, with the modification, which he thinks his own, of incising the sphincter in its lower half; he performed this iridotomy in about twenty cases. He gives no statistics, however, saying they are of value only to the operator. [This iridotomy, as a modification of Daviel's method, is already described by Beer ("Lehre der Augenkrankheiten," Vienna, 1792, vol. ii., p. 211), who says: "Even when the pupil is small and immovable, it generally dilates during the operation, and if this does not take place, it may be boldly enlarged by an incision."—*Reviewer.*] MARCKWORT.

ABADIE (232) recommends antiseptic precautions in cataract-extractions, combined with carbolic-acid spray, and eventually cauterization of the wound with the galvanocauter.

MARCKWORT.

PRIESTLEY-SMITH (242) finds by his investigations that "the growth of the lens does not cease with that of the rest of the body, but is continuous, unless morbid processes intervene, throughout the whole period of life." He gives a series of tables referring to the weight, volume, specific gravity, and diameters of the lenses examined. FITZGERALD.

According to TERSON (243), the extraction of cataract in patients affected with catarrh of the lachrymal sac is not attended with danger, if the tear-duct is syringed out with a four-per-cent. solution of boracic acid before the operation and every twelve hours afterward, the section made downward, and an antiseptic bandage applied, which is constantly irrigated with boracic acid.

MARCKWORT.

b.—VITREOUS BODY.

244. BAYER, FR. (Prague). Cases of visibility of Cloquet's canal in the eye. *Prager Zeitschr. f. Heilk.*, vol. iv., 1, p. 49.

245. LITTLE, W. S. Remains of the hyaloid artery attached

to the crystalline lens. Anæsthesia of the retina. *Trans. Amer. Ophth. Soc.*, 1882.

BAYER (244) reports three cases, the first of which presents the usual appearance; the second does not belong here; in the third there are diffuse retinitis and hyalitis in both eyes. In the left eye instead of the disk there is a white spot, with an indistinct lower margin and an irregular entrance of the blood-vessels. A grayish-blue membranous object, resembling a champagne glass, he believes to be the remnant of the fœtal vitreous, combined with a coloboma at the entrance of the optic nerve.

The condition described by LITTLE (245) existed in the right eye. In the left there were slight traces of the adventitia of the hyaloid close to the disk. This was likewise the case in a sister who came under observation later.

V.—RETINA AND FUNCTIONAL DISTURBANCES.

246. DENISSENKO, G. S. The changes in the eye in Bright's disease. *Med. Westnik.*, 1882, Nos. 49 and 50, and 1883, Nos. 2–11.

247. BAUDRY. De l'amblyopie unilatérale simulée. *Arch. d' ophth.*, vol. ii., 6, 1882, p. 496.

248. GROSSET, J. De l'amblyopie croisée et de l'hémianopsie dans les lésions cérébrales. *Rec. d' ophth.*, Mar., 1883.

249. MAUTHNER. Case of embolism of the central retinal artery. *Wien med. Blätter*, 1883, No. 9.

250. PAPILLIAN. Contribution à l'étude des tumeurs malignes de l'œil chez les enfants. *Thèse de Paris*, 1882.

251. SCHULIN, C. Embolism of the temporal branch of the central retinal artery, due to a fistula of the anus. *Northwestern Lancet*, Dec. 1, 1882.

252. MACKENZIE. Tortuosity of retinal vessels. *Trans. Ophth. Soc. of Unit. Kingd.*, iii., 1883, p. 101.

253. STORY. Anomalous distribution of the retinal arteries. *Trans. Ophth. Soc. of Unit. Kingd.*, iii., 1883, p. 102.

254. VIGOROUX. Amaurose hystérique dévelopée sous l'influence d'une impression morale et guérie en quarante-huit heures. *Paris méd.*, 1883, No. 43. No ophthalmoscopic changes.

255. WOLFE. On an operation for the cure of the detachment of the retina.

256. YVERT, A. Cas rare de rétinite albuminurique unilatérale observé chez un malade n'ayant qu'un seul rein situé du même coté et atteint de néphrite parenchymateuse. (Gros rein blanc.) *Rec. d' ophth.*, Mar., 1883.

DENISSENKO (246) examined every part of the eye in Bright's disease, and found no evidence of inflammation in any of the membranes; the cornea, sclera, conjunctiva, iris, optic nerve, retina, choroid, and ciliary body, however, were more or less infiltrated with albuminous liquid and œdematous, the interstices of the tissues being greatly distended or torn. The albuminous exudation may coagulate, and even fatty degeneration may set in. Only a few wandering cells could be found in the œdematous tissue. The visual disturbances are partly due to the œdema of the cornea, partly of the retina, though they may also be merely secondary phenomena, the result of ruptures, occlusion of the blood-vessels by an exudation, passive hyperæmia, extravasations, destruction of the rods and cones, etc. Sometimes thin coagulated deposits of albumen are found upon the lens. Detachment of the retina when it occurs is due to the exudation, while the dark spots in the choroid result from mechanical destruction of the pigment epithelium. The cause for this as well as the exudations in other parts of the body must be sought in the nervous system. The escape of the albuminous liquid into the tissues produces compression of the blood-vessels, and to it may be ascribed the hypertrophy of the heart and the extravasations of blood. All the ocular symptoms, except those due to destructive changes, may disappear when the patient is put upon nourishing diet and his general health is improved.

The author, therefore, proposes to drop the expression, "retinitis albuminurica" and substitute "ophthalmia Brightica or œdematosa" for it. The following ocular symptoms may lead to a diagnosis: absence of hyperæmia or even the presence of anæmia, small white streaks (not constant) at the sclero-corneal margin, more clearly visible with a lens, discoloration of the iris and sluggish reaction of the pupil, dulness of the iris and slight bulging, grayish appearance of the retina and albuminous deposits in it (the latter comparatively rare). The local symptoms do not require any special treatment. HIRSCHMANN.

YVERT (256) found retinitis albuminurica in the left eye only of a patient whose urine contained albumen; the patient was under observation for seventy-two days, when he died of nephritis

and its consequences. At the autopsy only one kidney was found, and this was severely affected with parenchymatous nephritis. Yvert quotes as analogous five cases published by Potain, in which there was anasarca of one side only, or at least more marked on one side, in consequence of contusion of a kidney, in explaining which Potain considers the sympathetic nerve of one side of great importance. Yvert also explains one-sided retinitis albuminurica by assuming an irritation of the sympathetic nerve of one side due to a unilateral affection of the kidney. MARCKWORT.

MAUTHNER (249) believes that embolism of the artery may be favorably affected in two different ways: by constant pressure upon the globe, when the embolus is still recent, in order to prevent it from being pushed farther into the artery, or, if it has already arrived there, to drive it forward and thus favor its escape to a more peripheric portion of the retina by diminishing the intraocular pressure; by incising the globe. Brilliant result in a case in which amblyopia had existed for seventeen hours.

SCHULIN (251) observed a case of embolism of the temporal branch of the retinal artery; the blood-vessel was reduced to the size of a thin thread. The corresponding part of the retina was pale, with a defect in the visual field of similar outline. There was a fistula of the anus. Six weeks later the branch in question was no longer visible, but the retina again had a reddish (?) hue, and the defect in the visual field began to disappear. Two months later sight had been completely restored. BURNETT.

WOLFE (255) punctured the sclera in five cases to facilitate the escape of the subretinal fluid; his method differs from that of Graefe and Hirschberg in so far only as he first detaches the conjunctiva at the point in question instead of incising it with the sclerotome. FERGUSON.

GROSSET (248) opposes the theory of simple semidecussation of the optic nerves, as well as that of Charcot, according to which some of the fibres cross in the chiasma, and the remainder in an unknown part of the brain, and bases his opposition on the clinical symptoms of some cases of cerebral affections which he cites from literature. He then sets up the following theory: 1. The inner fibres cross in the chiasma, while the outer ones continue directly. 2. The outer fibres cross behind the chiasma (perhaps in the region of the tubercula quadrigemina), thus making the decussation of all the fibres complete, and each capsula interna cerebri therefore contains all the fibres of the opposite eye. 3.

The outer fibres cross again beyond the capsula interna, before ending in the gyri, so that each occipital lobe contains the outer fibres of the eye of the same, and the inner fibres of the eye of the other side. MARCKWORT.

MACKENZIE (252) exhibited at the Ophthalmological Society of the United Kingdom a drawing of the fundus of the eye in a patient suffering from severe vesicular emphysema, œdema of the legs, and lividity. Retinal veins very tortuous, arteries slightly so. He said it might be ascribed to the venous obstruction consequent on the emphysema, though the condition was also sometimes seen in healthy eyes.

In STORY'S (253) case there was a curious arrangement of the superior nasal branch, which did not come from the disk, but appeared near its edge as a free loop whose two branches passed upward and inward, and horizontally inward respectively. The origin of this loop could not at first be found, but on dilatation of the pupil it was discovered that the lower branch communicated, by a large branch which presented some peculiar corkscrew bends, with the inferior artery above the point where the latter divided with the inferior nasal and inferior temporal arteries. S. gives a plate illustrating the appearance. FITZGERALD.

VI.—OPTIC NERVE.

257. EWETZKY. Atrophy of the optic nerve or glaucoma? *Med. Obosrenije*, May, 1883, pp. 374–390.

258. DE KEERSMÄKER. De l'atrophie du nerf optique observée chez plusieurs membres d'une famille. *Rec. d'ophth.*, April, 1883.

259. HIRSCHBERG. Affections of the optic nerve in malformation of the cranium. *C. f. A.*, vol. vii., p. 1.

260. VERON. Myxofibrome du nerf optique. *Rec. d'ophth.*, Jan., 1883.

261. ZACHER (Heidelberg). Choked disk in both eyes and perineuritis in hæmatoma of the dura mater. *Neurol. Centralbl.*, 1883, p. 125.

Based on three new cases and a recapitulation of four others previously reported, HIRSCHBERG (259) shows that *malformation of the skull*, either congenital or acquired very early in life, especially that form due to too early ossification of the sutures, is a rare but important condition accompanying *atrophy or inflamma-*

tion of the optic nerve. The first case was one of so-called tower-skull with early synostosis of the frontal bone and atrophy of both optic nerves after neuritis; the second case was one of a brachycephalous skull with the sagittal suture ossified, right-sided amblyopia, almost total left-sided amaurosis, and atrophy of the papilla; the third case was one of leptocephalus with atrophy of the optic nerves and amaurosis due to meningitis. The latter is generally the cause both of the visual disturbance and the malformation of the skull.

ZACHER (261) observed in a man of fifty-five with hereditary taint and melancholy, two weeks before death, slight paresis of the left side of the body and right-sided ptosis, which soon disappeared. Coma then set in, which continued until he died. Previously there was intensely marked *choked disk.* The autopsy revealed a *hæmatoma* of the *dura mater*, more developed on the right side than on the left; the sheaths of both optic nerves were distended; no liquid, however, escaped when a section was made. The contents proved to be the result of a previous chronic inflammation, which had produced adhesions between the inner and outer sheaths resembling a fine net-work. The compression manifested itself mainly in the veins, which were reduced to one half their size. The cause of this inflammation of the dural sheath could not be ascertained, whether it was due to the escape of cerebral liquid or direct spreading of the pachymeningitis.

EWETZKY (257) describes a case of steep and apparently total *excavation of the optic nerve* in both, and *loss of vision in one, eye* (fingers at 7,). Only the outer quadrant of the visual field of the amblyopic eye is preserved, and there is mydriasis. Both eyes are color-blind (with the better eye he only recognizes blue and yellow, with the poorer one only yellow). Has Robertson's pupillary phenomenon. Had diplopia several times; is unsteady with closed eyes. The patellar reflex is wanting. Had syphilis nineteen years ago. Diagnosis: spinal atrophy of the optic nerve from tabes; the excavation is an abnormally deep physiological one.

HIRSCHMANN.

KEERSMÄKER (258) reports in regard to a family in which axial atrophy of the optic nerve with central scotoma was hereditary. A toper, who frequently suffered from delirium tremens, had four sons and one daughter; all the sons and one son of the daughter suffered from more or less marked central scotoma and temporal atrophy of the papilla. MARCKWORT.

VERON (260) reports a case of myxofibroma of the optic nerve, which had existed for three and a half years and had been extirpated by Dr. Bouchez together with the eyeball. The tumor was egg-shaped, weighed twenty-two grms., and proved to be a myxoma changing to a myxofibroma. According to Veron the tumor began in the interfascicular tissue of the inner portion of the optic nerve, as the nerve fibres were much better preserved in the periphery. MARCKWORT.

VII.—INJURIES, FOREIGN BODIES (PARASITES).

262. DEBIERRE. Quatre cas de blessure de l'œil par coup de fleuret boutonné. *Journ. d'oc, et de chir.*, Mar., 1883.

263. EWETZKY, F. Cases of visual disturbance due to injuries of the skull. *Med. Obosrenije*, March, 1883, p. 374–390.

264. FANO. Observations de blessures de l'œil. *Four d'ocul. et de chir.*, Feb. 12, 1883.

265. GALEZOWSKI. Cysticerque sous-rétiniën. *Rec. d'ophth.* April, 1883.

266. GUERMONPREZ. Troubles nerveux consécutifs à une fracture du crane. *Gaz. des hôp.*, Feb., 13, 1883. Injury to the bones of the cranium and face of the left side, and amaurosis of the left eye with consecutive atrophy of the optic nerve and deafness on the same side.

267. HOMBURG, F. Statistics of cases of injury to the eye (Hirschberg's clinic). Berlin, Inaug. Diss., 1883, 116 p., 520 clinical histories.

268. SCELLINGO. Osservazioni di clinica oculistica. *Boll. d'ocul.*, vol. v., 5 gen., 1883. Six clinical histories of injuries to the eye. Nothing of importance.

269. SCHMIDT–RIMPLER. Contribution to the knowledge of some results of contusion of the globe. *Arch. f. Augenheilk*, vol. xii., 2, p. 135.

DEBIERRE (262) describes four cases of thrust against the eye with the button of a rapier. In two of them vision was preserved, but in the other two it was lost at once, while atrophy of the optic nerve followed. No ophthalmoscopic changes were visible before the atrophy of the nerve began. MARCKWORT.

FANO (264) reports two cases: (1) iridochoroiditis after gunshot wound—enucleation; (2) iris-hernia, due to an injury with a cutting instrument at the sclero-corneal margin; excision of the little tumor; good v. MARCKWORT.

Referring to his twelve cases in which a blow upon the eye with a blunt instrument had resulted partly in a reduction of vision, partly in the development of myopia and pupillary changes, SCHMIDT–RIMPLER (269) believes that these disturbances are not due to regular or irregular astigmatism of the lens, but to direct pathological changes in the retina, especially in the macula and its immediate surroundings, as shown by the ophthalmoscope in two cases. These changes may consist either in œdema, due to hemorrhage between the choroid and sclera, or to a strain of the tissue of the macula. The myopia is due to the traumatic irritation (? Ed.) of the ciliary muscle.

EWETZKY (263) reports the case of an epileptic boy of thirteen, who at the age of seven months was kicked on the back of the head by a horse. Neither externally nor with the ophthalmoscope can any thing abnormal be seen, though the eyes turn slightly to the left in fixing. The left half of the visual field is totally gone, the right is concentrically diminished and central vision reduced. Color-perception good in the whole field. The author thinks that the right visual centre was destroyed, and partly the left also, similar to the cases of Hugues, Schmidt–Rimpler, and Petrina.

GALEZOWSKI (265) incised the sclera in a case of *subretinal cysticercus* and extracted something with the forceps which he thought was the cysticercus, but which under the microscope proved to be an exudation with a small piece of retina and choroid. G. does not intend to make another attempt at extraction, but to destroy the cysticercus with galvano-cautery. MARCKWORT.

VIII.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

270. ABADIE. L'ataxie locomotrice est—elle d'origine syphilitique ? *Gaz. hebd. de méd. et de chir.*, December, 1882. He thinks it is not of syphilitic origin, and is therefore opposed to mercurials.

271. BLACHEZ. Traitement de la syphilis oculaire. *Gaz. hebd. de méd. et de pharm.*, May, 1882.

272. BOUCHUT. Diagnostic de la méningite et des maladies cérébrospinales par l'ophthalmoscope. *Rev. de cérébroscopie de l'année*, 1882 ; *Paris méd.*, 1883.

273. EPERON. Atrophie hémilatérale gauche de la face. (trophoneurose faciale). *Arch. d'ophth.*, vol. iii., No. 3.

274. HIRSCHBERG, J. On visual disturbances in progressive

paralysis. Psych. Ges., Berlin, Dec. 11, 1882. *Neurol. Centralbl.*, Jan. 11, 1883.

275. LANCERAUX. Syphilis cérébrale. Méningite syphilitique. Leç. faites en 1880 à l'hôp. de la Pitié et publ. par le Dr. A. Gautier. *Gaz. hebd. de méd. et de chir.*, January, 1883.

276. PARINAUD. Migraine ophthalmique au début d'une paralyse générale. *Arch. de neurol.*, Jan., 1883.

277. v. PFUNGEN, R (Meynert's clinic). Proof of a local cause of the disturbances of the movements of the eye in meningitis.—*Wien. med. Bl.*, 1883, 8–11.

288. RAMPOLDI. Sui rapporti fra le mallattie cutanee e l' organo visivo. *Ann. d' ottalm.*, vol. xi., 6.

289. REMY, E. Étude sur la tuberculose oculaire. *Thèse de Paris*, 1883.

According to BOUCHUT (272) there were among eighteen cases of tuberculous meningitis three with tubercles in the choroid; in all the cases there were marked changes in the optic nerve and posterior part of the eye. MARCKWORT.

According to REMY (289) pathological anatomy frequently fails us in making a differential diagnosis between tubercles and certain gummata of the eye, so that clinical symptoms must decide. MARCKWORT.

HIRSCHBERG'S (274) observations show that *atrophy of the optic nerve in progressive paralysis* is much more rare (4–5 per cent.) than is generally assumed. Sometimes there is also hemianopsia and a progressive central scotoma, a symptom generally overlooked, and discoloration of the papilla. He reports a very marked case, in which the field of vision resembled that of topers, though alcoholism could be excluded. The greenish-white papillæ and psychic and somatic disturbances proved the existence of progressive paralysis.

PARINAUD (276) saw chronic pachymeningitis follow several attacks of ophthalmic megrim in a man of thirty-four.

v. PFUNGEN (277) reports the clinical histories of eight cases to explain the ocular paralysis after meningitis; the inflammation of the meninges may produce paralysis: first, by an exudation at the base of the brain; second, by affecting the cerebral fissure (trochlearis, centre of co-ordination and association); and third, by affecting the cortical substance.

The patient of BLACHEZ (271), a nurse, was infected by a syphi-

litic child; iritis developed in the right eye, followed by a relapse seven months later; four weeks later irido-choroiditis set in both eyes. Cured by subcutaneous injections of mercurials.

MARCKWORT.

LANCERAUX (275) saw paralysis of the right oculomotor nerve and left-sided hemiplegia in a patient suffering from syphilis for a long time. MARCKWORT.

EPERON (273) examined a man of forty-one in Landolt's clinic suffering from atrophy of the left side of the face, which had begun in his eighth year after measles. There was hyperopia of 3 D in the left eye with V = $\frac{20}{200}$, field of fixation limited except downward. The ophthalmoscope showed a pale disk and atrophy of the choroid here and there. Right eye normal. The imperfect development of the left side of the face affects also the globe and muscles. v. MITTELSTÄDT.

RAMPOLDI (288) publishes a preliminary communication on diseases of the eye accompanying or following erysipelas, eczema, impetigo, psoriasis, and tinea favosa. DANTONE.

REPORT ON THE TWENTIETH ANNUAL MEETING OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY,

HELD AT THE GRAND HOTEL, CATSKILL MOUNTAINS, JULY 16 AND 17, 1884.

By H. KNAPP.

The Society was called to order at 10 A. M., and presided over by Dr. W. F. Norris, Vice-President, in the absence of Dr. Hy D. Noyes, President.

We shall give, as news, only short notices of the papers and discussions, referring our readers to the Transactions of the Society, of which the proper abstracts will appear in our systematic report upon the progress of ophthalmology.

The Secretary read a paper on *Iridectomy in Chronic Iritis*, by Hasket Derby, of Boston. After some introductory remarks, the writer reported in detail the case of a lady who had been treated for iritis for six years, both in Boston and New York. Relapses constantly occurred when mydriatics were left off. At times there was slight increase of tension, but never enough to constitute a glaucomatous condition. When she was comparatively well she made a journey to Europe, where she was persuaded to have an iridectomy performed in each eye. After her return the relapses of iritis occurred as before for eighteen months, then the tendency gradually disappeared. Dr. Derby did not ascribe the permanent recovery to the influence of the iridectomy, but to the final exhaustion of the inflammatory irritability, as is observed in so many cases, with or without iridectomy.

The discussion supported Dr. Derby's views.

The Secretary then read a paper by C. S. Bull, of New York, absent, on *Restoration of the Eyelid by Transplantation of Flap without a Pedicle, by Wolfe's method.* Reports of three cases:

CASE 1.—Complete ectropion of upper lid from burn. All the surrounding tissues cicatricial. Transplanted flap, $1\frac{1}{4}''$ long. Dressing left five days. Flap united, except at points of sutures, which suppurated. Result satisfactory. Six months of observation.

CASE 2.—Burn. Ectropium of upper lid. Lids stitched together. Flap, $1\frac{1}{2}''$ by $\frac{3}{4}''$, taken from forearm. United. Result good.

CASE 3.—Ectropium inferius from caries of orbit. Indrawn cicatrix, separated with tenotome; no benefit. Total excision of scar. The defect covered by transplanted skin; some sutures applied. Suppuration at points of suture; desquamation of epidermis in shreds. Sutures removed on sixth day. Result satisfactory.

B. E. FRYER of Leavenworth, Kansas, read a *Successful Case of Transplantation of a Skin-Flap without Pedicle, to Cover Eyelid, after removal of Epithelioma.* Eyelids closed with two silk sutures. Flap covered with gold-beater-skin plaster, adjusted with cotton pads steeped in boric-acid lotion, and then covered with another layer of the same plaster, protected with a cotton and flannel bandage. Patient immediately returned to his home, three miles off, where he removed the cotton and flannel bandage. The flap united. Result satisfactory. Time of observation, three months.

R. H. DERBY, of New York, read a paper on a *Case of Gangrene of the Lid, with Subsequent Restoration of Tissue without an Operation.*

Discussion of the three preceding papers:

T. R. POOLEY has seen remarkably good results when flaps sloughed after plastic operations.

GEO. C. HARLAN preserved the life of a gangrenescent transplanted flap by applying hot pads of cotton.

L. HOWE has followed up the previously published cases, especially his own, and found that the subsequent shrinkage of the flap continues for years, and is very considerable.

J. GREEN proposes to replace the sutures by small strips of gold-beater-skin plaster.

The further discussion dwelt upon the necessity of carefully cleansing the parts, perfect adaptation, and immobility.

GEO. C. HARLAN read a very entertaining case of *Hysterical Monocular Blindness with Mydriasis, Blepharospasm,* and paralytic

and spasmodic manifestations in the muscles of the face and other parts. The curious fact was that they rapidly disappeared upon the application of a Charcot magnet, but just as rapidly on the application of a wooden magnet with iron tips, perfectly imitating the Charcot magnet. ["Beware of counterfeits." ED.]

H. KNAPP, of New York, read a paper on "*Almost Sudden Loss of Sight in both Eyes under the Picture of Optic Neuritis.*" Two cases. In the first, sight was permanently lost in both eyes with the exception of a small island in the visual field on the nasal side close by the point of fixation, as far as it was possible to determine its locality. S in that island was $\frac{2}{L}$. The pupils were wide and immovable, and the optic discs became atrophic. In the second case, the total blindness with immobility of the pupils lasted only three days. Treatment: Profuse diaphoresis by salicylate of soda, and diarrhœa by calomel, about twenty-four stools in twenty-four hours. Gradual restoration of perfect sight in two months. Optic discs pale.

In the prolonged discussion following the paper many points in connection with optic neuritis were spoken of: its pathogenesis when no marked cerebral symptoms are present; its long duration in some cases without material changes either in its aspect or in sight; its presence without impairment of vision; its appearance on one side only, etc.

W. F. MORRIS, of Philadelphia, read two papers, (1) on *Hereditary Atrophy of the Optic Nerves.* Reports the history of a family of seven children. Parents had normal eyes. Several cousins affected. The disease was transmitted from the grandmother. Of twenty-two of her descendants fourteen were affected—nine males, five females. The amblyopia developed after puberty, in one earlier. He has watched one case for years. The affection began with slight neuritis, sight and color-perception still good. In a year central scotoma, with eccentric fixation. With the ophthalmoscope three stages of the disease can be distinguished: (a) slight neuritis with œdema; (b) œdema extended; (c) atrophy of the disc without marked changes in the calibre of the vessels. The sight in one of the patients has improved under hydrargyrum and strychnia treatment, in the others it has steadily diminished.

(2) Two cases of *Orbital Tumor*: the one a *myxo-sarcoma* in a child of four years, relapse after removal; the other an *osteoma*, slow growth, nine years. After enucleation of globe it was

removed with gouge and mallet. It consisted of cancellated bone. Thus far, five months, no relapse.

J. A. ANDREWS, of New York, read a paper on *Jequirity*. No new points were brought out, either by the paper or the discussion.

H. S. SCHELL, of Philadelphia, reported a *Case of Embolism of the Central Artery of the Retina*, in which the rosary-like appearance and the to-and-fro movements of the column of blood first described by Jäger were very well seen.

ARTHUR MATHEWSON, of Brooklyn, read a paper on a *Case Illustrating the Natural History of a Cataract.* After some years the cataract was spontaneously dislocated to the bottom of the vitreous chamber, at times producing glaucoma, which was temporarily benefited by eserine. Ultimately the eye had to be removed. The nucleus was very small and attached to the ciliary body.

GREEN related a similar case.

W. W. SEELY, read a paper entitled *Experiences in Refractive Cases.* Atropia is necessary, in the majority of cases, for a proper determination and correction of errors of refraction. Mixed astigmatism is rare ; he never saw it persist under atropia. Combination of myopia with insufficiency of the internal recti is frequent, and benefited by prisms. Myopia should always be determined with atropia and prisms.

Discussion.—MITTENDORF finds mixed astigmatism frequent. He often orders prisms, not always with the basis laterally, but at times up or down.

L. WEBSTER FOX, of Philadelphia, contributed the *Clinical History of a Case of Sympathetic Ophthalmia*, distinguished by the rapid destruction of the eye.

J. A. LIPPINCOTT, of Pittsburg, O., read a paper on *Two Cases of Orbital Abscess.* In the first the abscess appeared in the lid soon after erysipelas. Later, exophthalmus. Vision and interior of eye normal. Pus found near inner canthus. Recovery in five weeks. The other orbit had to be operated on—incision—two days after the first. Recovery. In the second case offensive pus was liberated by an incision. Later a spontaneous opening discharged healthy pus.

THEOBALD related a case of orbital abscess which ended in recovery, though for some time the patient alleged that he could not see.

C. J. KIPP, of Newark, N. J., read a paper on *Abscess of the Frontal Sinus.*

CASE 1.—Patient complained of frontal headache for years. Hard swelling along the whole superior and part of the inner-upper orbital margins. At the operation it proved to be a bony shell, containing a great quantity of pus. Patient died of lobar, not of pyæmic, pneumonia. The frontal, ethmoidal, and sphenoidal cells were united into one large cavity.

CASE 2.—In the course of a year, abundant discharge of offensive pus through the nose several times. Then a tumor formed at the inner-upper corner of the orbit. By external pressure it was emptied into the nose. Up to the present time, a year later, it has not reappeared. Violent blowing of the nose forces air into the frontal sinus, and crepitation is felt in the adjacent skin.

L. HOWE, of Buffalo, read a paper on the *Changes in the Interior of the Eye Immediately after Death.* He made successive ophthalmoscopic examinations of a dying man. The main feature was the gradual bleaching of the fundus.

THEOBALD, of Baltimore, reported a case of *Glioma of the Retina* with secondary tumors of the skull. He presented photographs illustrating the case.

W. S. LITTLE, of Philadelphia, read a paper on the system of examining the personnel, 40,000 men, employed by the Pennsylvania Railway Company. The examinations for color-blindness and acuteness of sight and hearing have been conducted according to W. Thompson's method for several years and have given entire satisfaction.

GEO. C. HARLAN, of Philadelphia. Two cases of *Swelling of the Optic Disc*, about $\frac{1}{15}$ H with ophthalmoscope, the picture of optic neuritis, but possibly only a congenital elevation. Patient a young man ; myopic ; S normal.

The second case a girl of fifteen years. Occasional dizziness and nausea. Neuro-retinitis stationary. S good. Cerebral symptoms cured with atropine and glasses.

RISLEY and MITTENDORF mention cases of neuro-retinitis with perfect sight.

GUST. HAY, of Boston, communicated two cases of *Conical Cornea* in which he had used *Rählmann' hyperbolic lenses* with good results.

WADSWORTH, of Boston, related a case of *Myxœdema with Atrophy of the Optic Nerves ;* calibre of arteries and veins diminished.

W. wants to put the case on record, though the optic-nerve atrophy may be a coincidence. He has not found it present in other cases of myxœdema, nor has Dr. LITTLE.

T. R. POOLEY read a paper on a case of *Acute One-sided Dacryoadenitis.* It followed the loss of the other eye by blennorrhoic conjunctivitis. Recovery without suppuration.

MITTENDORF, of New York, read a paper on *Polycoria.* Three cases. The first, the most remarkable, showed five pupils in one eye, one central and four peripheric. The latter were conical in shape with their bases at the periphery like a coloboma. The central only dilated by atropia. Accommodation perfect. No other defect in eye.

SEELY has seen five or six members of one family affected with polycoria. They all became blind from choroiditis later in life.

E. HUTCHISON, of Utica, N. Y., presented photographs of a patient who had sustained a very severe injury of the lids of one eye from a blast. The palpebral fissure was narrowed, the lids so much drawn toward the angle of the lower jaw that vision was practically made impossible. Cure by loosening and raising the lids.

J. AUB, of Cincinnati, O., reported four cases of *Removal of Foreign Bodies from the Vitreous.*

CASE 1.—Piece of steel penetrating at margin of cornea without wounding the lens. Extraction with Bradford's magnet through enlarged wound on fifth day. A second piece situated deeper in the eye removed later likewise with the magnet. Recovery. S $\frac{20}{30}$. Last seen ten months later.

CASE 2.—Extraction of a piece of steel, $\frac{1}{3}''$ by $\frac{1}{16}''$, through opening in sclera. S $\frac{20}{30}$. Jäger 1; fundus clear. Two years under observation.

CASE 3.—Cornea perforated, iris prolapsed. Iridectomy on tenth day. Foreign body seen on background two months after injury. Later, after violent exertion the eye became irritated. The foreign body had left its place and was seen on the lower ciliary region. Permanent cure after successful extraction with magnet through an opening in the sclera.

CASE 4.—Piece of iron injured cornea and lens. Lens gradually absorbed. Cyclitis some months later. Unsuccessful attempt to extract the foreign body with magnet. Enucleation. Foreign body encapsuled in ciliary region.

In a lengthy discussion several members of the Society commu-

nicated their experience on the same subject. The permanent magnet, on account of its weakness and unreliability, was considered inferior to the electro-magnet.

H. KNAPP reported a successful case of extraction of a piece of steel from the vitreous, though it could not be seen on account of opacity of the lens.

H. KNAPP and J. GREEN reported cases of foreign bodies which, after having penetrated the eyeball, were, after enucleation, found encapsuled at the posterior surface of the sclerotic.

S. D. RISLEY, of Philadelphia, reported a case of *Hypermetropic Astigmatism Passing for Myopia.* S = $\frac{20}{20}$, with and without glasses. Nervous, even mental, symptoms for years. Intense headache every day. Finally cured by atropia and astigmatic glasses combined with prisms.

In the afternoon session of the first day a paper of Dr. DYER on a *New Perimeter*, was read and illustrated by a figure. Further, Dr. A. ALT, of St. Louis, exhibited macroscopic and microscopic specimens of a rabbit's eye in which he had produced a croupous internal inflammation by injecting an infusion of jequirity into the vitreous. In the other eye a moderate croupous exudation was found in the anterior chamber and upon the optic nerve. He stated that interstitial neuritis was present in both optic nerves, and pronounced the affection in the second eye to be *experimental sympathetic ophthalmia.*

We may fairly say that the meeting was very successful. The papers were numerous, and all were of interest. The discussions were animated and instructive. The social intercourse of the members was amicable, even cordial. One passing cloud of an ethical nature cannot fail to exert a beneficial influence on the future of the Society. The Committee on Admission did not report the name of a gentleman whose scientific and literary prominence is acknowledged by all. The committee laid his name over for the next year on account of the doubtful origin of some small newspaper items in which he was favorably mentioned. The member of the Society who had proposed him said that he appreciated the committee's vigilance and endeavor to keep bad men out of the Society, but in the present case their objections rested on suspicion only, and if newspaper paragraphs that could be construed as advertising were sufficient to exclude a gentleman from this Society, there would be no member left. On the whole, he thought that the ethical side of a candidate had, thus

far, been too much considered, whereas scientific attainments were never mentioned. At the close of the meeting Dr. J. Green, the Chairman of the Committee on Admission, proposed the following amendment to the By-laws: "*As members of the Society can be admitted only such physicians as have been engaged in the practice of ophthalmology for five years, have shown evidence of scientific attainments, and have conducted themselves in conformity with the ethical rules of the Society.*" This amendment was unanimously adopted.

For the ensuing year Dr. NORRIS, of Philadelphia, was elected President, and Dr. WADSWORTH, of Boston, Secretary, Dr. R. H. DERBY having resigned the latter position, which he had filled with so much aptitude and benefit to the Society for ten years. The determination of the place of meeting in 1885 was left to the choice of the Secretary.

MISCELLANEOUS NOTES.

We take pleasure in noticing a **new American text-book**: A Treatise on Ophthalmology for the General Practitioner, by Adolf Alt, M.D., St. Louis. J. H. Chambers & Co., 1884, pp. xvi., 244. Price $3.00.

This work, the author informs us in his preface, is not designed for the specialist, but "is intended to serve as a guide for the general practitioner as to when he may conscientiously take upon himself the responsibility of dealing with an eye affection and when he had better not do so." The book should therefore be judged by this standard which the author has set up for himself; and looked at from this standpoint it appears to us to fulfil its purpose as well as any work of the kind can do. The doctrines, so far as they are taught, are sound and up to the latest discoveries in our science, and the general practitioner who goes to it for knowledge will be correctly informed. The faults of the book are those inseparable from its class. Ophthalmology has become so distinct as a science and art that even its principles cannot be taught in a few short lessons—and most manuals give both too much and too little. They deal with questions with which the general practitioner should have nothing to do—as the extraction of cataract for example,—while refraction and ophthalmoscopy, with which his work is most intimately associated, are not dwelt upon at sufficient length to be of any practical value to him. Dr. Alt,

has, however, performed his difficult task with judgment, and the practitioner will find his little book a safe guide.

We regret that greater care has not been exercised in the proof-reading, and that some of the illustrations are so rough and sketchy. These detract much from the appearance of the volume which is otherwise very neat in its get-up. S. M. B.

A little book—336 duodecimo pages, flexible binding—has just appeared, and, though not treating of the science or practice of medicine, will be of great service to such physicians as are learning German. Its title is **"Medical German,"** a manual designed to aid physicians in their intercourse with German patients and in reading medical publications in the German language. By SOLOMON DEUTSCH, A.M., Ph.D. New York: J. H. Vail & Co.

The first part contains the medical terms, systematically arranged in different chapters—for instance, the bones, cartilages, and ligaments, the vascular system, the five senses, appetite and thirst, general aspect of patient, surgical instruments, remedies and expedients, etc., etc., giving the translations of all the words (5,633) used in that connection. The second part consists of seventy-five conversations, taking up the most important subjects, with questions and answers between physician and patient—for instance, on diseases of the eye, six pages. At the end there is an alphabetical index of all the German words, and another of all the English words, used in the volume, very convenient for reference in reading and translating. The importance of the German language for physicians, and for oculists especially, being so very great, we thought we would render a service to many of our readers in bringing the above very useful and reliable little book to their notice. H. K.

The next meeting of the **American Medical Association** will be held in New Orleans, La., the last Tuesday in April, 1885. Section of Ophthalmology, Otology, and Larynology, Dr. J. A. WHITE, of Richmond, Va., President, Dr. EUGENE SMITH, of Detroit, Mich., Secretary.

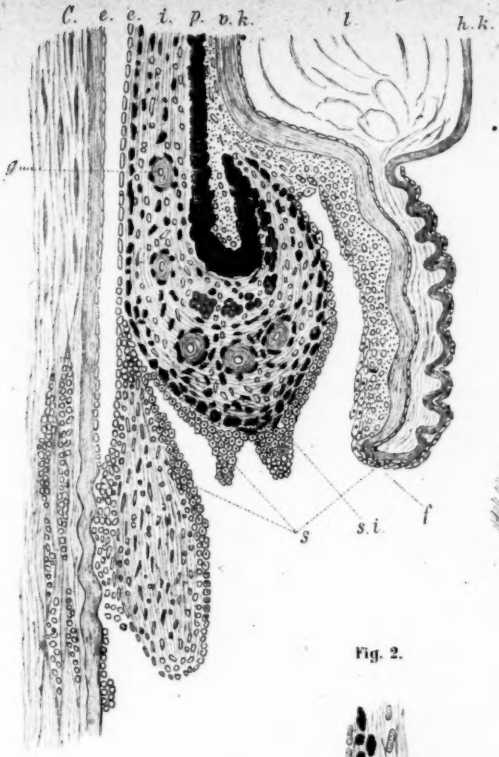
The meeting of the **Ophthalmological Society in Heidelberg** will be held Sept. 14th to 16th, 1884.

In the new National University at Prague, Bohemia, Dr. SCHOEBEL will occupy the chair of Ophthalmology; and for the place of Prof. Hasner, resigned, Profs. O. Becker, Fuchs, and Sattler have been nominated. Prof. O. Becker declining, received the title of *Geheimer Hofrath*, in Heidelberg.

It is proposed to organize in Boston a **New England Ophthalmological Society,** similar to the one for twenty years in successful operation in New York City. Meetings to be held at the houses of members, on the first Tuesday of each month, from November to April, inclusive, at 8 P. M. Adjournment to take place at 10 P.M., and to be followed by a simple collation. Organizing Committee: Hasket Derby, B. Joy Jeffries, Oliver F. Wadsworth.

ERRATA.—In Dr. W. W. Seely's communication, p. 167 of this volume, it is stated twice that he uses a solution of bichloride of mercury gr. i. ad ℥ i. of water; it should read:—gr. i. ad O i. (pint), about 1 : 8000.

Fig. 1.



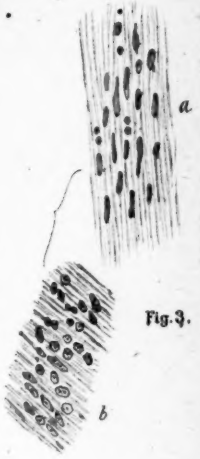


Fig. 3.

Fig. 2.

Fig. 4.

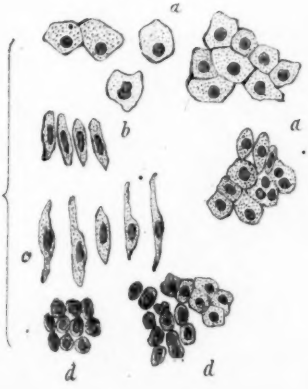


Fig. 5.

Verlag v. J. F. Bergmann, Wiesbaden.

Lith. Anst. v. F. Wirtz, Darmstadt.

Fig. 1.

Lith. Anst. v. Werner & Winter, Frankfurt a/M.

Fig. 2.

Fig. 5.

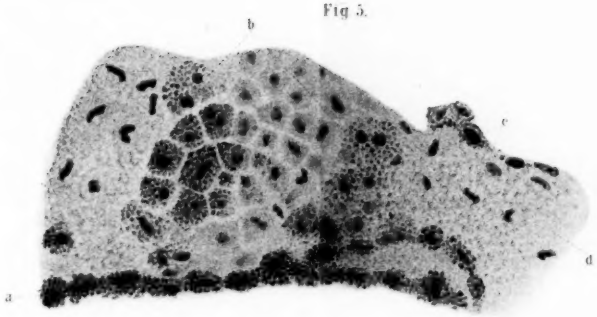


Fig. 3.

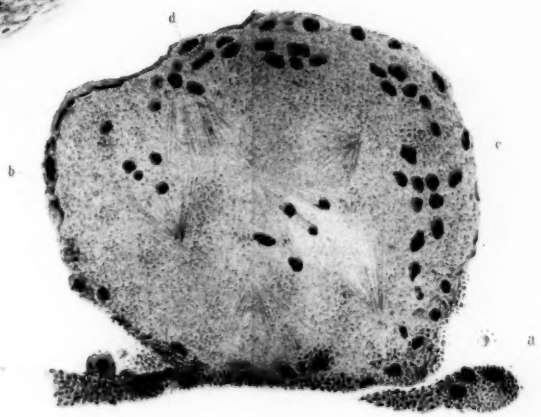
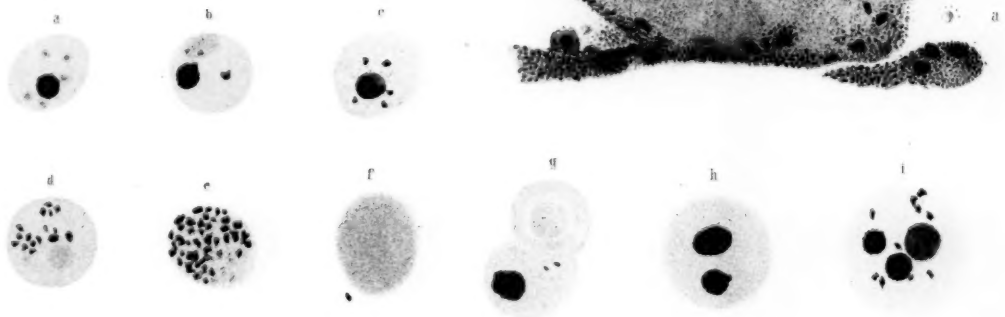



Fig. 4.